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CONTENTS

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AETIOLOGY AND CLINICAL ASPECTS OF CARIES AND ITS TREATMENT *

ROBERT HARRIS, M.D.S.

AETIOLOGY.

In this discussion we are concerned only with the enamel. The well-known clinical picture of the carious lesion seen so frequently is the macroscopic evidence of a process which has commenced some considerable time before and then persisted for a period in microscopic dimensions.

The generally accepted opinion concerning the mechanism is that caries is caused by acids resulting from the action of certain micro-organisms on carbohydrates. It is characterized by a decalcification of the inorganic contents of the enamel, accompanied by the disintegration of the organic substance.

It is primarily initiated in areas of the tooth where food debris collects and, therefore, pits and fissures and the proximal surfaces of the teeth are the selected areas. Apart from these morphologically-grouped regions, certain structural defects play an important role in the causation: these are lamellae which run from the tooth surface towards the dentine and hypoplastic enamel. The lamellae in a vital tooth in association with the bacterial plaque seems to provide a mechanism for the initiation of acid production in intimate contact with enamel, and the hypoplastic structure provides additional defects for food lodgement.

We know that the structure of the enamel is approximately 95% inorganic material and the balance organic, and that it is covered at eruption and for a short time thereafter with an organic cubicle which may form some protection against acid material, but within a very short time this is damaged either by attrition or dissolution so that it cannot play a protective role for any length of time. The interprismatic substance is that portion of the enamel which contains the greatest portion of the organic constituents; some investigators (Gottlieb, Pincus) believe that this provides the initial pathway for the

*Delivered at the Plenary Session, The Cause and Prevention of Dental Caries, of the Twelfth Australian Dental Congress.

destruction and that, once this material is disintegrated, then the enamel rods fall away and cavitation commences.

The first visible evidence of caries on an intact enamel surface is a change in the optical properties of the enamel: by reflected light the area appears white and by transmitted light there is a darkening; the microscopic picture changes so that the rods appear more definite and the cross striations become more noticeable.

Acid is produced from the carbohydrates substances after they have been acted upon by bacterial enzymes. The enzymes have their origin from the oral flora and, therefore, any organism or combination of organisms which is capable of maintaining an adequate acid potential sufficient to decalcify enamel will be capable of initiating dental caries.

The oral flora contains a number of organisms: lactobacilli, aciduric streptococci, diphtheroids, yeasts, staphylococci and others, capable of producing this acid potential *in vitro*. The lactobacillus has been found to show a significant increase in numbers with the initiation of caries. Decalcification is dependent upon the degree of the acidity and the time that the acid is in contact with the enamel: it has been shown that an acid potential of pH 5.0 is sufficient to decalcify the inorganic structure, although circumstances might vary considerably in the conditions found in the mouth.

Without more detailed discussion, it can be stated that the conditions must be suitable, both in regard to pabulum (substrate) and enzyme system on the one hand and tooth structure and morphology on the other, to complete the vicious circle which leads to a carious lesion.

Restating this concept in another way, we may say that the effect of caries is made possible by circumstances which retain acid in contact with the tooth. Such circumstances are:—

1. The anatomic characteristics of the tooth.
2. The position and arrangements of the teeth.
3. The presence of prosthetic appliances or restorations.
4. The dental plaque, an organic mass containing multitudes of organisms which is firmly adherent to the tooth.
5. The physical structure of the enamel.
6. The requisite substrate.

Any concept of the process must satisfy the condition enforced by the physical facts just enunciated.

There are, however, numbers of other theories postulated which are based on other concepts such as those used by the geneticists and nutritionists as well as a mechanistic theory: however, despite evidence which their advocates have collected, the most satisfactory theory supported by the greatest volume of evidence which is sufficient to enable one to draw certain conclusions, is Miller's chemico-parasitic theory.

Taking the conditions enunciated as one group, the other main factor playing a vital part in the process is the saliva which continually bathes the enamel. It may, by dilution and by its buffering capacity, reduce the effect of acid production. Some authorities have suggested that individual resistance

to caries is due to inhibitory substances in the saliva, although evidence in support of this is not as yet conclusive.

The relation of systemic conditions to the caries process has not clearly been established and further studies of this problem are required. In some cases individuals in poor nutritional status have a low degree of caries activity.

Considerable data have been collected and presented on the relationship of nutritional status and caries, particularly in relation to calcium and phosphorus content, vitamin D and the physical nature of the diet. Klatsky and others have attempted to link the physical nature of food with the prevalence or prevention of caries, but analysis of the evidence reveals that no carefully controlled study has been made which establishes definitely a relationship between the detergent or abrasive quality of the diet and the prevalence of dental caries in man, although recently Neumann has sought to show that this is the case and has claimed that the effect of sugars can be eliminated by the use of tough detergent diets. Unfortunately, in the studies that have been made there are too many uncontrolled factors, and conclusions have been drawn from the works of others without due cognizance of the limitations noted by the original investigator.

A summary at this stage would seem to indicate clearly that the situation which determines whether or not the carious lesion develops depends upon the interrelation of all the preceding variables, and no single factor may be construed as the general cause of all dental caries.

Clinical Aspects.

On the basis of the concepts enunciated, it seems that we can control dental caries by decreasing the forces which tend to cause caries, or by increasing the resistance of the tooth to attack. Any method which we use, however, must not be drastic, since the disease is not fatal; secondly, it should be harmless and, thirdly, because of the universal prevalence of the disease, simplicity of procedure is most necessary. Perhaps of, by far, the greatest importance is its acceptability by the public and its availability to the public.

Fluorination of drinking water, once it is established as an acceptable method of control, would appear to satisfy most of these demands.

Other methods now available can be used as supplementary procedures, but it should be remembered that fluorination of drinking water has yet to be proved—the evidence is considerable from epidemiological studies that there are wide variations in the amount of caries in areas having naturally occurring fluorides and non-fluoride areas—it is an assumption, as yet, that such data will appear to support the addition of fluorides.

One important point must not be overlooked by the profession: with the evidence being steadily compiled indicating that certain therapeutic agents are useful as preventives, the possibilities of abuse should be guarded against. The composition and degree of purity of test materials are important factors. Too many commercial products tend to fall into such a category.

Methods of Control.

The main efforts today are directed towards reducing the incidence of caries by directing control of the bacterial and chemical environment of the teeth and can be summarised into categories as:

1. Reduction of the amount of fermentable materials entering the oral cavity, i.e., diet control.

2. Removal of fermentable substances before they are converted into acid, i.e., oral hygiene.
3. Inactivation of the enzyme system responsible for fermentation, i.e., enzyme inhibitors such as 2 methyl-1: 4 naphthoquinone (Menadione or so-called Vitamin K), zephiran and urea, and in a minor way sodium fluoride perhaps.
4. Neutralization of the acids before damage is done, i.e., by the use of alkaline agents.
5. Production of a tooth tissue less vulnerable to attack, i.e., by the use of sodium fluoride and tooth impregnation methods.
6. Alteration of the bacterial flora of the mouth, i.e., using ammonium salts and certain antibiotics such as penicillin and chlorophyll.

Taking these six factors together with the mechanical treatment of the carious lesions, we have reduced the problem to a reasonable dimension for our conception.

(1) The first and, in theory, readily controllable factor is a reduction in the amount of fermentable materials in the diet. By the institution of the appropriate restrictions in the amount of refined carbohydrates, dramatic reductions in the lactobacillus counts have been obtained and this count—the lactobacillus index—is indicative of a reduction in caries activity. Some authorities contend that, if the diet is nutritionally adequate, then the carbohydrate content is not significant; however, if a dietary regimen is to be instituted, some obvious control is essential to gain and maintain the patients' co-operation. It is, therefore, important to institute measures which make the patient fully aware that his co-operation is essential and that there are means readily available to evaluate the standard of co-operation. Two tests can be used and should be demanded by the dentist as an aid:

(a) The lactobacillus count which can be undertaken by any bacteriology laboratory—this can be implemented at the first examination and, unless in an immune mouth, will always be positive and is directly related to the caries activity. The patient is then instructed as to the dietary regimen and subsequent tests are made at intervals; this will provide a direct indicator of the degree of control—has another valuable use, since the lactobacillus count can predict caries formation in patients who at the time of examination have no clinical evidence of caries. Used in this way, the practitioner is armed with a valuable aid in caries prevention. In such cases, with patient co-operation, it is possible to forestall the carbohydrate degradation and ultimate acid formation.

(b) The Snyder Test is an indicator of the presence of other acid-producing organisms in saliva, which would not be evaluated in the lactobacillus count. It has a slight advantage, from the patient's angle, over the lactobacillus count, since the test depends upon the change in colour of the medium used from green (brown-cresol green) to yellow. The more rapid the change, the greater the concentration of acid-producing organisms, and this fact can be impressed visibly on the patient's mind.

(2) Removal of fermentable substances before they are converted into acid has long been considered an important factor—a clean tooth never decays—but the impracticability of this procedure can readily be demon-

strated and, therefore, its value is largely reduced, since it has been shown that in the appropriate environment acid production develops within a few minutes of the presence of the carbohydrate and in the course of a meal the buffering effect of the saliva is largely eliminated. The question has been raised as to why sugars in fruit juices do not have the same effect as refined sugars and carbohydrates. Any effect from acid production is dependent upon the presence or absence of the bacterial plaque. It has been noted that erosion is present in areas where there are no plaques—the mouths of patients who drink quantities of lemon or orange juice. Where the plaque is present on a vital tooth, erosion does not occur but caries may commence; where it is absent, erosion is likely to occur simply by solution of the enamel by the acid content of the fluid.

Another problem arises as to why, in the presence of plaques in certain positions and the presence of sugars or other refined carbohydrates, caries does not appear; it has been suggested that lamellae offer a mechanism for the institution of an osmotic pressure drawing sugar beneath the plaque into intimate contact with the enamel. Enzyme action follows and acid production develops undisturbed.

Prophylaxis of itself is not sufficient to reduce caries, since it has been shown that plaques will grow back over the tooth surface within one to four days of the prophylaxis. In any case, prophylaxis is only likely to be effectively carried out in accessible regions of the tooth. Careful toothbrushing immediately after meals does remove plaque material from accessible surfaces.

Dentifrices will contribute to the prevention of plaque formation, particularly if they have some abrasive, but the brushing technique is more important than the dentifrice. The use of dibasic ammonium phosphate and urea in the dentifrice will reduce the lactobacillus count, but there is insufficient evidence that penicillin and other antibiotics and fluorides have any effect.

Although prophylaxis and toothbrushing can be recommended, "for numerous reasons the evidence at present that such measures are effective in the prevention of dental caries is limited and at present inadequate."

(3) Prevention of this enzyme action may be possible and investigations upon the effect of Menadione (2 methyl-1: 4 naphthoquinone), incorrectly referred to as vitamin K, have shown some promise in the control of bacterial growth. Iodoacetic acid has been shown to be an enzyme inhibitor and similar action is thought possible as explaining some of the effects of sodium fluoride. Zephiran and urea also have some action as enzyme inhibitors.

(4) The use of alkaline agents would appear to be ineffective if acid production depends upon the plaque as a protective medium, since the alkali would have to penetrate the plaque in suffiicient concentration to neutralize the acids formed.

(5) Sodium fluoride appears to be capable of causing changes in the structure of the enamel depending upon the concentration of the fluoride and the time of contact. This effect may be demonstrated by the presence of fluorosis where the fluoride content of drinking water is above 1.5 ppm. and by reduced caries incidence in areas where the content is around 1-1.5 ppm., demonstrated particularly in the mouths of those patients who have lived in the areas from birth. Topical application with 2.0% solution also appears to

be effective if applied at the 3, 7, 10, 13 age groups, provided that at each series the initial application is preceded by a prophylaxis.

Recently, the use of silver nitrate and zinc chloride has been suggested on the basis that the caries action is a proteolytic one and either of these agents applied will precipitate the protein content of the inter-prismatic substance and lamellae and, therefore, block the organic pathway.

Topical application of sodium fluoride as a therapy is predicated on the basis of the absorption by bone meal of sodium fluoride from aqueous solutions: it is noted that enamel and bone meal are hydroxy apatite calcium salts and, therefore, a change apparently occurs in the superficial enamel layers. Prophylaxis should be carried out prior to the first application of any series, but whether this should be carried out at subsequent visits in each series is very doubtful, since the effect of the fluoride will be only on the superficial layers and this may be polished away. Increase in hardness has been demonstrated.

(6) Alteration of the bacterial flora of the mouth has been suggested and is the basis of the ammonium ion dentifrice measures currently in use.

Lactobacillus acidophilus counts are reduced, following the use of a tooth powder containing dibasic ammonium phosphate and urea. The ammonium ion inhibits the lactobacillus which allows the entrenchment and growth of Bacterium aerogenes. Enzymes produced by this latter organism break down amino-acids and liberate ammonia which creates a difficulty for the re-establishment of the lactobacillus. A cycle is created which, if maintained by regular application of the dentifrice, prevents re-establishment of the Lactobacillus acidophilus.

Finally, the practitioner has still to rely upon the mechanical reparative processes for the treatment and, to a large extent, the control of dental caries. Its value as an aid for the prevention of dental caries should not be overlooked for, certainly, once the cavity has commenced its extension is not likely to be prevented by any other means. One great difficulty in evaluating a caries treatment process, whether preventive or control, is that the presence of caries detected clinically is often difficult and uncertain, whereas, as was stressed in the beginning, the presence of the cavity occurs in the later stages.

Early orthodontic procedures may also be advantageous where it appears that malocclusion will follow and, in the young child, adequate restorative treatment should be instituted in order to maintain function and develop correctly aligned arches.

In applying preventive measures, the private practitioner should remember that individual cases will arise in which results cannot be obtained comparable with those demonstrated in large surveys. For example, topical application of sodium fluoride has been demonstrated to reduce caries incidence by 40%; it does not necessarily follow that we can expect such a reduction in

every case. The private practitioners should, therefore, be aware of this and should advise their patients that they can expect no more than certain results from various procedures and, in some cases, the result may be disappointing.

Spectacular results are not to be expected from dietary procedures unless they are designed to reduce the intake of refined carbohydrates.

In conclusion, it seems that best results can be expected from the application of the following procedures: adequate and regular dental care, topical sodium fluoride therapy, restricted intake of refined carbohydrates with appropriate controls and adequate oral hygiene.

BACTERIOLOGICAL PROBLEMS OF DENTAL CARIES*

N. E. GOLDSWORTHY, M.B., B.S. (SYD.), PH.D. (CANTAB.), D.P.H., D.T.M. & H.

SUMMARY.

(a) To understand a disease we need first a knowledge of the anatomy and physiology of the organ or tissue involved. At present our knowledge of enamel is inadequate.

Therefore we cannot dogmatise upon the pathology or the pathogenesis of dental caries: we must be prepared to consider alternatives.

(b) Two broad groups of chemical substances are present in enamel:—

(1) inorganic salts,

(2) protein.

Opposing schools of thought emphasise the importance of one or the other, and in so doing focus our attention on two different mechanisms for the destruction of the enamel:—

I. Those who support Miller's views stress the action of acid on the inorganic components of enamel;

II. Those who oppose Miller's views are predominantly concerned with the action of proteolytic forces.

In either case it is a reasonable assumption that bacteria through their metabolic activities are the exciting causes of dental caries.

(c) The outstanding bacteriological problem is to establish a body of sound knowledge of the oral flora and its activities under the various conditions found in the mouth. Some of the relevant difficulties are mentioned.

(d) Prevention must be based on a knowledge of aetiology and at present this is deficient.

We have evidence to support an assumption that bacteria are the chief exciting causes.

Therefore prevention will include anti-bacterial measures, among which the most important appears to lie in the field of dietetics.

BACTERIOLOGICAL PROBLEMS.

Any consideration of a pathological state must clearly be based on a sound knowledge of the structure, composition and function, i.e., anatomy and physiology, of the tissue or organ concerned. It is only too obvious from a perusal of the literature that this necessity sets irksome limits to our discussion of that commonest of pathological states and processes known to our patients as decay of the teeth. We have not so far been able to agree on some very pertinent aspects of the anatomy and physiology of dental enamel. The result is that perplexing diversity of emphasis on one or other of the real or imaginary structures in enamel and the erection thereon of a correspondingly rationalised hypothesis of the pathogenesis of the disease. Too often is research into this and other conditions converted into a hunt for new evidence, observational or experimental, in support of a point of view, rather than an attempt to find the truth. Admittedly we generally have points of view and take sides, so to speak, for the too open mind is apt to be condemned as colourless, irresolute, undiscriminating. It is, however, contrary to scientific principles to draw conclusions from inadequate data. A rigorous examination of the literature would in all probability prompt the purist to discard most if

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not all of the so-called evidence for the aetiological role of even that most-studied oral inhabitant, the Lactobacillus—and indeed it is futile and dishonest to shut our eyes to the fact that bacteriological studies of dental caries have mainly been made on mouths containing several clinically obvious lesions or on teeth similarly affected. Unfortunately the earliest stages or the initiation of the lesion cannot as yet be detected. We tend to forget that dissolution or removal of substance or structure may proceed at submicroscopic levels, as in chemical reactions, where ions, atoms and molecules are the reacting units. Yet it is at and just before this initial and submicroscopic stage of the process of caries that we should seek the exciting causes. For the time being, then, we must be content with what is in effect indirect evidence and that, too, based on an incomplete knowledge of the structure of the tissue concerned.

The title of this contribution to our discussions is "Bacteriological problems of dental caries." This, of course, must be considered in relation to the general title of the discussion for this Plenary Session, namely, "The cause and prevention of dental caries." The phrase "Bacteriological problems of dental caries" implies in part at least that bacteria are concerned in either the cause or prevention of the disease or in both. We know that outside the animal body the tooth can be the most resistant of all the body's tissues. Archaeological finds are sometimes limited to a piece of partly disintegrated bone and one or more teeth apparently in a state of perfect preservation. On the other hand we know that the tooth within the mouth can be rapidly destroyed by caries. The outstanding difference is clearly one of environment, using that word in its broadest sense, and bacteria are surely part of the environment of the tooth.

Broad agreement seems possible on the chemistry and histology of enamel to the point of admitting that both inorganic substance (prisms) and organic substance (prism sheaths) are present at the surface and there form a kind of mosaic or tessellated pattern. There is thus exposed (I) naked but very slightly proteinised apatite (or calcium phosphate for the purpose of our argument) and (II) naked but heavily calcified keratinous protein. Opposing schools of thought emphasise either (I) or (II) as the tissue-element primarily attacked.

I. Those workers who support Miller's views regard the calcified element as the object of primary attack, so that the prisms are more likely to suffer than the prism sheaths. Because calcium salts are distributed throughout all the component parts of enamel, it is difficult to conceive of the destruction of the prisms or even the calcified sheaths in the manner characteristic of caries by any other mechanism than that of attack by acid. There is ample and incontrovertible evidence that bacteria of many species can form organic acids from a variety of substances. There is likewise evidence that similar acids are formed in the mouth in certain circumstances and that no mechanism for forming these acids in the mouth can be detected other than the processes of bacterial metabolism. As a basis then for this part of our discussion we shall accept the available evidence as support for the assumption that acidogenic bacteria are concerned in the aetiology of caries.

Among workers who support Miller's views there are two main groups: (1) those who hold that one or other specific micro-organism is the exciting cause and (2) those who hold that two or more micro-organisms can be the exciting cause.

(1) Of all the organisms severally claimed to be the specific cause of caries the Lactobacillus (so-called) has been most studied and it is a depressing reflection that ease of study may be one reason for this. Yet it is by no means universally accepted that the Lactobacillus possesses the importance so frequently attributed to it: other workers have consistently pressed the claims of various streptococci to first place. One cannot help feeling that the only justifiable interpretation of the apparently conflicting evidence is that more than one species of organism can cause (initiate and/or promote) caries insofar as they can produce sufficient acid in specific environments and suitable circumstances to decalcify enamel. It is clear that we shall have to explain why the Lactobacillus cannot be isolated from some patients' mouths although their teeth show obviously active caries. My own teaching for more than ten years has been that it is probable that there are many agents and combinations of agents and circumstances which will enable the production and accumulation of acid in sufficient concentrations to initiate and promote caries.

(2) While, then, there is reason to suggest that several species of organisms may individually initiate and promote caries, more interesting is the possible combination of bacteria for team work, a subject far too long neglected in the study of medical and dental bacteriology. In this connection there is evidence that the Lactobacillus and certain yeast-like organisms may work in symbiotic and synergistic combination more effectively than could either alone. Unfortunately, however, probably owing to technical difficulties, very little work has been done to extend this kind of observation.

II. Those workers who do not accept Miller's views have mostly chosen to emphasise the organic or protein elements in the enamel as the object of the primary attack. Pincus and Gottlieb represent this group of workers, although they severally propose rather different mechanisms of attack upon the enamel. Destruction of organic material as such (the prism sheaths) will necessitate the production of proteolytic enzymes in suitable amounts and circumstances. No exception can be taken to their views because of this requirement: various micro-organisms from the mouth have been shown to possess the power to attack proteins. Nevertheless it is not easy to understand how the enamel can be eventually disintegrated without the aid of acid, because to the best of our knowledge even the protein in it is heavily impregnated with calcium salts.

In considering these opposing concepts, let me remind you here of my opening remarks that any satisfactory concept of the pathogenesis of caries must be based upon our knowledge of the structure and chemistry of enamel. In view of our uncertainty of these, then, it is hardly proper to dogmatise concerning the validity or otherwise of these opposing views (I and II above) although our limited albeit voluminous evidence (especially that on the dietetic control of caries) would seem best to support Miller's concept.

Cox has advanced the not unattractive view that carious lesions have an individuality depending upon such factors as position on the tooth, position of the tooth and the differences in the flora at different parts of the mouth and in different mouths.

The difficulties, therefore, of establishing the claims of any one or more species to be considered as the exciting cause of dental caries will be obvious.

The outstanding bacteriological problem, then, is to define the conditions under which bacterial species either singly or in combination can produce in the mouth metabolites or enzymes capable of destroying enamel. Obviously the first step must be the building up of a body of accurate and detailed knowledge of the whole flora of the mouth. This is a stupendous task, so terrifying in its immensity that we may well be forgiven for avoiding it.

Not only is the oral flora at any given time extremely numerous and varied but also it is in a state of constant flux. We might then suppose that it is impossible to define the oral flora and, to some extent, this is true but progress has indeed already been made in this direction. We recognise, for instance, that there is a basic flora which includes Streptococci of the viridans and gamma groups and that for certain individuals other organisms are fairly constant members of the flora; for example, Candida or other yeast-like organisms occur in approximately 30 per cent. of young adults and their occurrence is constant in any one individual over a long period of time. The same perhaps might be said of the Lactobacillus: some persons habitually harbour it, others do not.

At the moment we are faced with the fact that no *single* species of bacterium can *always* be isolated from the mouth of persons with active dental caries and that within the carious lesion one or another of several organisms of diverse morphology have been repeatedly described as occupying the most advanced zones of the diseased tissues of the carious tooth. Once again, if we accept this conflicting evidence as valid, we can only conclude that caries is indeed caused or (more accurately) promoted by more than one species of bacterium. Clearly any definition of the oral flora and its environment must take into account the state of the teeth themselves; in other words immune and susceptible subjects must be compared. Unfortunately work along these lines has not shown any invariable difference between the two groups of subjects despite the impressive literature now collected round the Lactobacillus. This would seem to indicate once more (a) that more than one species can be concerned, either separately or in combination, or (b) that the enamel-destroying capabilities of any one or more species, singly or combined, is dependent upon environmental factors such as quantity, quality and frequency of supply of suitable substrate; this question of substrate may be considered from two opposite aspects, that is to say the supply of substrate may either promote the formation of the destructive metabolites or enzymes or on the other hand it may promote the production of substances capable of neutralizing or inactivating these destructive substances.

Prevention can be discussed only in the most general terms since its nature will depend upon the aetiology of the disease. Assuming that a case for bacterial action has been established, we may proceed along one or more of the following lines:—

(a) The most obvious method of attacking offending organisms is to remove them, as for instance is attempted but never attained by oral hygiene. Few, however, would on the basis of present knowledge recommend the discontinuance of oral hygiene. There is, for instance, the well-known observation of Fosdick, that when oral hygiene is energetically and conscientiously practised immediately after eating it can effectively limit the initiation of new lesions. On the other hand we must admit that, as usually practised by the

so-called "average" person, oral hygiene is little more than a gesture justifiable mainly on aesthetic grounds. Never before has so much oral hygiene (so-called) been practised among civilised peoples, yet never before has the incidence of dental caries been so high among these same civilised peoples.

(b) We may attempt to interfere with the metabolism of the organisms by attacking their enzymes either (i) by the application of specific and recognised enzyme poisons, for example fluorine or (ii) by the application of antibiotic mechanisms, for example by implanting harmless organisms recognised to be antibiotic towards suspected aetiological agents (Aerobacter versus Lactobacillus) or by the use of antibiotic products (penicillin). While there is experimental evidence for the partial success of (i) in animals, it is at present doubtful whether this measure is applicable on an effective scale to human beings. Also the establishment in the mouth of organisms which might prevent other bacteria (such as Lactobacillus) from initiating or promoting lesions is by no means easy, unless assistance is given by

(c) the use of selected diets. Diets should be chosen for their ability not only (i) to promote the growth and activities of the implanted organism but also (ii) to depress those of any particular aetiological agent, or if possible (iii) to act in both ways.

If we disregard for the moment the associated economic and psychological problems, dietetic measures for the control of the oral flora are the most promising of all the caries-control mechanisms directed against oral bacteria; for example, it has been clearly shown that a diminution in the intake of sugar and refined carbohydrates generally will result (at least under certain conditions of living) in a fall in the lactobacillus count and in the incidence of new lesions.

This dietetic approach to the problem of control of dental caries can, then, legitimately be regarded as bacteriological in nature and should be energetically made, despite the obvious difficulties which attend it.

NUTRITIONAL ASPECTS OF THE PROBLEMS OF DENTAL CARIES *

CECIL HEARMAN, D.D.Sc. (MELB.).

INTRODUCTION.

It has been stated that 80% of the diseases to which the human flesh is heir are due to errors in nutrition. This opinion may, or may not, be an exaggeration, but it can be stated, without fear of contradiction, that as far as dental caries is concerned, it is entirely due to dietetic errors. The mechanism of the carious process is, as yet, not fully understood, but the actual causative factors are known. There seems little doubt that the initial lesion is the result of the bacterial decomposition of fermentable carbohydrates in the external environment of the tooth. What happens after the lesion is established is pathologically significant but, strictly speaking, it is hardly pertinent to a discussion on the prevention of the lesion itself. However, with the advent of the proteolytic theory, a new aspect of the problem has presented itself. As a result of more recent research, a number of investigators believe that a tooth which contains a relatively high content of organic material, as a result of incomplete maturation, is much more susceptible to carious attack. The question naturally arises whether these incompletely calcified areas could be prevented by nutritional means, and so render the dentition less vulnerable to attack. This possibility will be discussed in this contribution, although it represents a somewhat indirect approach to the problem of total prevention, which could only be achieved by removing the actual causative factors.

It would be a comparatively simple matter to prevent dental caries if fermentable carbohydrates could be eliminated from the conventional dietary. This, of course, is neither practical nor possible. Unfortunately, the foodstuffs, which are responsible for the disease, have come to be considered a necessary and delectable content of all civilized dietaries, and the advocacy of their restriction is not acceptable in general to sufferers from dental caries. This means that research workers have had to concentrate on alternative preventive methods, which would be acceptable to the general public. It seems that some method will have to be determined of preventing the formation of the dental plaque, or inhibiting the enzymic degradation of fermentable carbohydrates, within the plaque. Alternatively, we might find some way of rendering the tooth itself more resistant to the series of destructive reactions which follow when organic acids are concentrated on its external surface. The first-mentioned methods represent a dietary problem, the second is thought by many to be a nutritional one.

The terms, diet and nutrition, are often loosely applied, but it is a first essential to this discussion that the difference to their meaning and application be clearly understood. The term nutrition refers to the intake of foods and their subsequent metabolic change, within the system, to supply the needs of the tissues and cells and other bodily requirements. Diet, on the other hand, refers to our food as it comes off the plate. Prevention of dental caries by *nutritional* means thus involves a consideration of *systemic* factors, whilst

*Delivered at the Plenary Session, The Cause and Prevention of Dental Caries, of the Twelfth Australian Dental Congress.

prevention by *dietary* means concerns the purely local effect of foods when taken into the mouth. We will deal with each aspect of this problem separately.

THE PREVENTION OF DENTAL CARIES BY NUTRITIONAL MEANS.

Theoretically, there are two ways by which we could employ the nutritional factor to prevent dental caries. They are:—

1. Application during the formative period, in an endeavour to credit the offspring with teeth, so fully calcified, that they would be resistant to carious attack.
2. Application after tooth eruption, in an attempt to improve the oral defensive mechanism, by increasing the calcium content and buffering potential of the saliva.

The first method is a very popular one with the laity, who firmly believe that resistance to dental caries is dependant on the possession of "good, strong, well-calcified teeth." This belief became more strongly engendered as the result of the investigation carried out by Lady May Mellanby about 15 years ago. Lady Mellanby claimed that hyphoplastic enamel structure was due to calcium and vitamin D deficiencies during the pre-natal period, and that such hypoplastic teeth were more caries susceptible. This conclusion was arrived at as a result of a series of experiments on animals, who were fed on grossly deficient diets, and a large-scale investigation on children, to whom these theories were applied. The Mellanby experiments have since been subjected to much adverse criticism, which has shown that her methods were fallacious, and her conclusions unjustified. Despite this, many members of the medical and dental professions prescribe calcium and vitamin D supplements to the diet of the expectant mother in the belief that these additions will produce teeth in the offspring which will be more resistant to caries. This practice is now so widespread and general that every dental and medical practitioner should know whether these beliefs are possible of fulfilment and whether they are based on sound, physiological principles.

Before we can consider the full implications of the theory crediting tooth structure with resistance to the carious attack, it is necessary to understand the present-day conception of the mechanism of the carious lesion. First of all, there is no doubt that the lesion commences on the external enamel surface, and there is also no doubt that a concentration of organic acids on this surface initiates the process. Enamel consists of a relatively small amount of organic binding material, in the form of a matrix, and a much larger proportion of calcified material in the form of hydroxyapatite crystals, which are precipitated into the matrix. The carious destruction of these two entirely dissimilar tissues thus involves a two-phase process, which consists of a dissolution of the organic material by bacterial enzymes and a concomitant chemical decalcification of the calcified material. The concentration of acids on the external surface can be regarded as a type of "trigger" mechanism which sets the more complicated resultant reactions alight. If protein hydrolysis is the *first* of these reactions then a tooth, having all of its organic pathways completely obliterated, would be theoretically caries-resistant. The question whether this is capable of practicable attainment is one which concerns every practising dentist. Let us, therefore, review the evidence regarding this possibility.

Because the carious attack is directed against the external layer of enamel, it is obvious that this is where we would have to build a defensive barrier. Schour has shown that the amount of enamel present at birth can be determined by the neo-natal ring, which is an incremental line dividing the pre-natally formed from the post-natally formed tooth structure. This incremental line indicates a deficient calcification of the enamel, resulting from the disturbance which occurs in the nutrition of the foetus when its nutrition is changed from the medium of the placenta to the stage when it derives its food supply from the mouth following birth. The *significance* of the neo-natal line is that it shows that relatively little calcification of the enamel takes place in the deciduous dentition before birth and that the major and outer portions of the enamel are formed during the six months pre-eruptive period after birth. This knowledge, alone, effectively disposes of the principle of *pre-natal* calcium feeding as a means of producing an outer layer of enamel which could be more resistant to caries.

The next possibility to be considered is whether the same objective could be achieved by nutritional supplements, administered during the neo- and post-natal periods. To achieve complete maturation and calcification of *all* the organic material in the enamel it would be necessary to obliterate the lamellae, the enamel tufts, the rod sheaths, and the organic matrix of the rods, and the intercementing substance. With the possible exception of the lamellae, all of the remaining structures are *normal* constituents of the enamel, and are placed there to serve normal physiological purposes. The enamel tufts bind the dentinal and enamel matrix together at the dentino-enamel junction, whilst the organic sheaths and matrix are a necessary and integral part of the enamel structure, providing it with a very necessary degree of resilience against the stresses of mastication. It is, therefore, neither likely nor desirable that these normal constituents could be dispensed with. According to Gottlieb the lamellae are formed by tensions set up within the organic matrix during the maturation process. They are caused by the deposition of calcium and the withdrawal of water and protein during the process of calcification. If this is true, then hypercalcification should produce greater tensions and, consequently, greater numbers of lamellae! Finally, it still remains to be shown histologically that the organic structures in enamel can be obliterated by nutritional supplements administered during the developmental period. Although this practice has had widespread clinical application during the last quarter of a century, no one seems to have reported definite evidence of improved tooth structure in consequence.

The next question concerns the possibility of altering the anatomical configuration of the tooth, to reduce the number of stagnation areas. Schour and Massler, as a result of their studies on tooth development, have shown that "the view that the depth of occlusal pits and fissures can be controlled by nutritional means is erroneous." These structural defects are genetically determined and are the result of the fusion of appositional growth centres established during tooth development at the relatively early stage of morphodifferentiation.

These authors also emphasise that enamel hypoplasias are rarely caused by nutritional deficiencies but are the result of permanent cell injuries. Such injuries as rubella, and the exanthematous fevers affect the ameloblast cells,

and the developing enamel, and once hypoplasias and hypocalcifications are formed they are permanent and cannot be corrected by any dietary regimen. For example, fluorosis has a toxic effect on the ameloblasts and produces hypocalcification of the enamel, but it has no corresponding effect on the dentine. On the other hand, deficiencies of calcium and vitamin D *do* affect developing dentine but *do not* affect the calcification of the enamel.

We can summarise by saying that, so far, there is no biological evidence that we can improve the quality or structure of enamel by nutritional means so as to render it caries-resistant. All of the evidence indicates that the calcification of enamel is well-safeguarded before birth unless the formative cells are subjected to toxic or infective interference. Day and Taylor have shown that pre-natal calcium deficiencies, severe enough to cause osteo-malacia in the mother, and rickets in the offspring, do *not* adversely affect the calcification of the enamel or pre-dispose the teeth to dental caries.

This view is also supported clinically, for I am unaware of any published evidence that people suffering from rampant caries show concomitant symptoms of systemic calcium deficiencies. Rickets, evidence of such a deficiency, is almost unknown in Australia, but the incidence of caries among pre-school children has been shown to be as high as 97%.

The best evidence against the theory that caries can be controlled by nutritional means is furnished by the results of the post-war dental survey of Italian children by Schour and Massler. As a result of the war, most of these children suffered from gross nutritional deficiencies during their formative period. These deficiencies were severe enough to cause general malnutrition, anaemia, and rickets. Yet these same children had seven times less caries than well-fed comparable groups of children living in the United States.

So much for the structural theory which, after 15 years of clinical trial, has produced very disappointing results. The next objective which might be attained by nutritional means offers a little more encouragement.

Increasing the Calcium Content and Buffering Potential of the Saliva:

Boyd and Drain, amongst others, have shown that an increased buffering potential of the saliva may be achieved by feeding on alkaline ash diet. Such a diet includes an abundance of fruits, fresh, raw vegetables, salads and dairy products. It is hard to assess the protective value of the increased buffering potential, because such a diet includes many detergent and few caries-causative foods.

Gottlieb claims that saliva which has a relatively high calcium content may provide an additional defence against caries by precipitating a deposition of calcium salts into exposed lamellae. This action, he says, not only prevents invasion along these pathways, but also prevents the dental plaque from gaining an attachment to the tooth.

The Dietary Factor as applied to the External Environment of the Tooth:

No one seriously disputes the fact that the degradation of fermentable sugars and starches into acids, which are concentrated on the tooth surface, initiates the carious process. Of all the foods eaten by man only the cooked starches and sugars are capable of undergoing enzymic degradation to acids in the mouth. They are thus the sole caries-causative foodstuffs. To initiate caries the starches must be of a sticky, tenacious consistency because their relatively slow fermentative action requires prolonged and close approxima-

tion to the tooth surface before the acids can reach sufficient concentration to cause damage. Such foods as cakes, biscuits, bread and jam, and most patent breakfast foods fall into this category. Unless the starches are mixed with sugar they possess very little lodgeability. Consequently, such starches as spaghetti and macaroni are relatively harmless. Sugars, to be caries-causative, must be taken in fairly strong solutions so that they develop sufficient osmotic pressure to cause deep penetration of the dental plaque. Weak solutions of sugar, such as those occurring in natural fruits, remain on the surface of the plaque where they are neutralized by the saliva or the plaque itself. Concentrated sugars, in the form of sweets and candies, may develop an osmotic pressure of as high as 60 atmospheres. Under these conditions sugar solutions are drawn deep into the plaque and, where lamellae are present, into the tooth itself. At the same time water is withdrawn from the plaque and its buffering capacity is broken. The conditions are now present for the enzymic degradation of the contained sugars to organic acids although the extent and exact nature of the reaction is further governed by the bacterial population of the dental plaque. Stephan believes that, when lactobacilli predominate in the plaques, they halt the degradation process at the lactic and pyruvic acid stage, and permit a more prolonged accumulation of these acids. In the presence of other organisms a relatively quicker oxidation of these acids takes place to carbon dioxide and water. In view of this knowledge it is only natural that we should ask the question—are these dentally dangerous foods necessary to the general health of the patient? The answer is decidedly in the negative. Both sugar and white flour are highly refined and chemically bleached extracts of the natural product, and it is most unlikely that nature intended their use in such concentrated form and quantity. The consumption of sugar in Australia has now risen to the really alarming figure of 131 lbs. per head per annum. At the present time we eat about seven times as much sugar as people living in Italy and Spain, and it would be interesting, indeed, to study the comparative dental pictures.

There is no *real* need for sugar in the diet. Contrary to general belief, it does not provide energy, unless the vitamins Thiamine, Niacine, and Riboflavin are present in sufficient quantity to ensure its complete metabolism. Excessive quantities of sugar in the diet result in a deficiency of these vitamins. The normal individual can obtain all his glycogen requirements from the starches in whole-grain cereals and potatoes, the sugars in natural fruits and some vegetables and from lactose in milk, plus honey in small quantities. It is well to remember that cane sugar does not contain fats, proteins, minerals, vitamins or roughage. It is a dead food, containing neither protective nor body-building elements. On the other hand, it provides the substrate for enzyme action, it promotes the growth of lactobacilli, it depresses the growth of the opposing ammonifying organisms, it perverts appetite, it decreases the intake of protective foods, it increases the lodgeability of starchy foodstuffs and it diffuses readily into the dental plaque. The sum total of these reactions may be expressed in the simple statement: sugar rots teeth.

A plain, simple, balanced dietary, which bans entirely sugar, biscuits, sweets, cakes and jam, will practically eliminate dental caries and at the same time it will promote a well above normal standard of good health. No better example of this fact is available than the one provided by the Bailey experiment with institutional children at Bowral in this State. This experiment was

started in 1942 with the result that 86 children now have fewer cavities between them than in one jaw of the average child.

We can summarize the whole of this contribution in one sentence: as far as the prevention of dental caries is concerned it is more important to know what to leave out of the dietary rather than to worry about what should be put into it.

OPPORTUNITY FOR PRACTICE AT GRETA.

It has been brought to the notice of the Newcastle and Hunter River District Division of the Australian Dental Association, New South Wales Branch, that an opportunity exists for the establishment of a successful dental practice near and in the New Australian Centre at Greta. Further information may be obtained from the Secretary of the Division, Post Office Box 302, Newcastle.

SELF-CURING RESIN — ITS USES AND LIMITATIONS *

P. R. Rheuben, D.D.S. (Minn.).

The silicate cements, or so-called synthetic porcelains, have filled a necessary place in dentistry for many years. We are all aware of their shortcomings and many of us have waited patiently for the day when we could throw them out of the window. I, personally, still have my silicates in the cabinet but once or twice I have had my hand on the window catch.

When a self-curing resin was introduced about two years ago for use in the laboratory in making "cold-curing" repairs on acrylic dentures, my interest was very much aroused. I had long before intimated to a chemist that the use of a catalyst which would polymerize methyl methacrylate at mouth or room temperature would be of great value to dentistry. With this new laboratory material there seemed little doubt but that such a catalyst or activator was in use and must be in either powder or liquid form, or both. By taking the liquid of one of the cold-curing acrylics and adding it to the powder which is used in making acrylic jackets and bridges, it was found that polymerization took place very rapidly and the resultant mass, when allowed to harden under pressure, had the appearance and properties of a desirable filling material.

This mixture formed the basis of some of our earlier fillings and these are still in the mouth today.

I do not know to whom credit is due for the development of these materials, but I am led to believe that a self-curing resin was in use in Germany at the close of the war and that a catalyst therefrom has served as a basis for the development of our present self-curing resins. My correspondence with manufacturers has not been enlightening, and in some repects the boost given to their own product and alleged research thereon has been too much for me to swallow.

Naturally, in the beginning the possibility of pulp injury was not overlooked. However, it could be reasoned that, once polymerization had taken place, the resultant acrylic would be almost inert. While these details were receiving consideration, it was already being established elsewhere that the effect on the pulp was more less negligible. These facts have been verified by competent authorities on behalf of more than one manufacturer and the details are readily available.

Brief Chemical and Physical Properties.

Polymerization.

The process of polymerization of methyl methacrylate for denture work has been described in dental literature many times during the last twelve years; this polymerization for self-curing resin can be regarded as essentially the same.

Whereas in denture resins we may have an accelerator in the powder which with heat and pressure brings about polymerization, in self-curing resins we probably have an accelerator in the powder plus an activator or catalyst in the liquid, the resultant mixture of powder and liquid generating a certain amount of heat to aid its own polymerization.

*Read at the Twelfth Australian Dental Congress.

The pressure necessary to produce a good dense resin is just as important in a self-curing resin as it is in a denture resin.

One manufacturer has made the following observation with respect to polymerization, which should give us food for thought:

> "The polymerization of methyl methacrylate whether the processing be carried out at an elevated temperature or at a low temperature is initiated by the decomposition of an organic peroxide (generally benzoyl peroxide). This decomposition produces a free radicle. It is this free radicle which attacks the monomer molecules, thereby initiating the polymerization process. In the resins which are identified as thermal curing resins, this decomposition of the peroxide is accomplished by heat. In the case of the self-curing resins, the decomposition of the benzoyl peroxide is achieved by reaction with a tertiary amine.
>
> "Since amines generally show some discolouration upon oxidation, there will in all self-curing resins be some tendency to discolour. As is obvious, the discoloration will be dependent upon the specific amine employed and the concentration of amine and peroxide necessary to give the desired curing characteristics."

Shrinkage.

There is no doubt about shrinkage taking place during polymerization A "constant pressure" technique has been devised to offset this to some extent.

As further compensation, I believe that a certain amount of expansion may take place afterwards, due to absorption of water by acrylic.

Solvents.

Generally speaking, any solvent which will attack denture resin will attack self-curing resin. For this reason the cavity and any instrument or matrix, etc., should be dry and have no trace of chloroform, alcohol, phenol, lubricant grease or impression compound.

Phenol in very dilute form will cause self-curing resin to become rubbery; for this reason I abstain from the use of phenol in the toilet of the cavity.

Impression compound will leave a stain and otherwise ruin the surface; hence it is necessary to tinfoil any surface which will be in contact with the filling area.

It is only natural that with a new material the great majority of dentists will be more interested in how to use it than in details of scientific tests which will interest but few.

The purpose of this paper, therefore, will be to give an account of techniques for the classes of cavities where success can be reasonably assured. In doing so, I would like to point out that much is being learned from experience as we go along, and it is possible that entirely new techniques may be developed between the time of writing of this paper and its presentation.

The Manipulation of the Material.

Those with considerable experience in handling acrylic in the laboratory have a head start over all others. There is no substitute for experience in this field.

The wetness of the mix, the characteristics of the mixture at the time when it is ready to pack, the packing, pressing and finishing—all follow a definite similarity to the handling of denture acrylic. To make up for any lack of experience in this regard, it would be wise to make several mixes of the material about to be used and study its characteristics. The material so used, however, need not be wasted. It may be used, before it has got too doughy, to pack into a small copper band and allowed to set under finger pressure for a

few minutes. It will be noted that there is a definite rise in temperature and, after the material has set, the copper may be stripped away and the resultant cylindrical piece of material may be slightly flattened by grinding and polishing one end. This will serve as an excellent colour guide and thus give an accurate verification of colour for each batch of material.

Mixing.

It would be wise to follow the directions given by the manufacturer of the particular product being used. There are certain characteristics, however, which may be observed in all materials during their manipulation. Probably all require that enough liquid be present to wet all the powder without being sloppy. It will be found from experience that the best results are obtained with a certain minimum of liquid, but not so little that the mix will be too granular. Our personal preference is to use as small a jar as possible, make the mix by allowing the liquid just to saturate the powder very slowly, give it a quick mix and then close the jar.

Do not open the jar until you think the mixture is nearly ready. The consistency should be that of an adhesive dough; in most cases it will be found advantageous to start packing just before the "smooth dough" stage is reached, so as to take advantage of whatever adhesiveness there is and not be too late in applying pressure.

Colour.

Most manufacturers put out a wide range of colours. If it is not desired to use all of these, select say three dentine or cervical colours varying from pale yellow or almost white to a dark yellowish brown; two yellowish greys or greyish yellows will serve for most incisal colours. A bottle of grey and a bottle of blue will also be useful as modifiers for some of the unusual colours. When two or more colours are to be used, thoroughly mix the dry powders before adding any liquid and then again after the mixture is wet.

The tendency in the beginning is to select a colour that is too light, due probably to the fact that acrylic is very translucent and the colour guide has greater bulk than the proposed filling.

Setting Time.

The fact that dentists have been used to materials that could be mixed after the cavity has been prepared, and then inserted immediately, is no doubt responsible for the manufacturers decreasing the setting time. Whether this is achieved by increasing the accelerator or not, I do not know, but the fineness of the powder would no doubt be a contributing factor. At least one manufacturer recommends the use of a cavity liner.

I do not believe it a disadvantage to have a resin which is slow to jell after mixing. It is purely a matter of making the mix five or ten minutes, as the case may be, before you expect your cavity preparation to be completed.

Polymerization is considerably affected by climatic conditions; temperature is probably foremost, with altitude and humidity no doubt playing their part also. For this reason, study the behaviour of the material in your own locality in both winter and summer.

Pressure for at least ten minutes during the setting period is an absolute necessity. This cannot be emphasized too strongly for, if it is possible to exert

considerable pressure and maintain it during this time, there will be fewer failures.

Indications for Use.

Primarily the use of a self-curing resin is for aesthetic reasons; therefore, there is no point in using it in posterior teeth where it is not visible. The gold inlay and alloy fillings cannot be easily ousted from the high position they have held as regards durability.

1. In cases where one would normally use a silicate filling or in many cases a porcelain inlay, it can safely be assumed that the use of a self-curing resin is indicated. It is especially useful in Class V cavities in anterior teeth.
2. For use as a temporary anterior jacket crown and possibly as a permanent jacket also.
3. For use as a temporary anterior bridge to replace a broken porcelain bridge or to make a temporary repair on a porcelain bridge while a new one is constructed.
4. Repairing, replacing or constructing in crown and bridgework that part which originally may have been constructed in porcelain.
5. Setting of acrylic crown and bridgework.

Contra-Indications.

1. Most cavities wherein the filling would be subject to considerable attrition or stress of mastication, mainly Class I and II.
2. Proximal cavities in anterior teeth which cover an extensive area without providing sufficient undercut or retention.
3. Class IV cavities which involve a large amount of incisal edge or an unfavourable bite.

Cavity Preparation.

The principles of cavity preparation should be borne in mind continuously but emphasis should be placed on retention.

We are handling a material which is entirely different from all others so far. It is resilient and horn-like, and when set is easily peeled from smooth surfaces; hence, the use of square margins and the undercutting of the cavity are of the greatest importance.

To secure undercuts, the wheel bur is particularly useful, especially in Class V cavities. The No. 14 wheel is about the best average size. In Classes III and IV, undercuts may be made with a No. ½ round bur, especially in the point angles. Suitable hatchet chisels may then be used to emphasise the undercuts further, especially towards the incisal in Class III cavities.

In those Class IV cavities which are deemed suitable, the No. ½ bur may be used to undercut the incisal edge working towards the opposite angle.

The Insertion and Compression of the Filling.

As already pointed out, the consistency of the material at its insertion should be that of a dough which has not lost its adhesiveness.

It should be packed as quickly as possible, overfilling the cavity so that, when pressure is applied, a well-condensed filling will result.

The exact method of applying pressure will vary for each class of cavity, and it is the successful application of this pressure which distinguishes success from failure.

There are now on the market several varieties of clamps which have been designed to maintain pressure on the filling during its setting. In spite of all

these, I still favour the use of an Ivory No. 4 Matrix retainer, converted by removing the wedges and substituting pins. In addition, a "hatch" clamp, a pair of haemostats (artery forceps), copper bands and a few other articles will complete the armamentarium. A separator with points ground may be used instead of the No. 4 Matrix retainer.

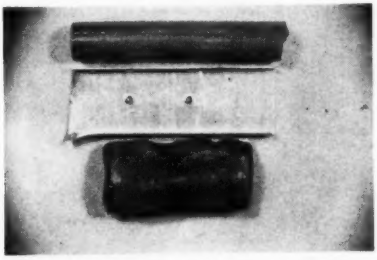


Fig. 1.—Showing stick compound and aluminium plate and compound fused on to aluminium.

CLASS V (SMOOTH SURFACE CAVITIES).

These have been placed first because they are the easiest to manage and because they constitute the type of cavity wherein the indication is strongest for the use of self-curing resin.

(1) *Where the cavity does not extend under the free margin of the gum.*

Take a piece of aluminium plate similar to that used by some manufacturers for carding artificial teeth, and fuse on to it some stick compound. (Fig. 1.) Have several like this made up for future use.

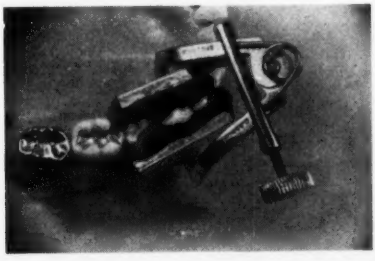


Fig. 2.—Pressing a typical Class V cavity, using old separator as clamp.

Soften the compound and take a labial impression of the tooth to be filled, as well as one tooth on either side to aid positioning. Cut from the impression any mark made by the cavity and also any pronounced ridge which may be present at the gingival. Now tinfoil beyond the area which will cover the cavity, making sure that no cracks appear over the filling area, as the compound may seep through and stain the filling.

A similar metal-backed impression is made for the lingual which will not need tinfoiling.

The cavity should now be prepared and the mix made. When the cavity has been filled generously, the metal-backed impressions are seated and the clamp is applied (Fig. 2.), taking about a minute to get full pressure. At least ten minutes should elapse before the clamp is removed.

The use of compound, rather than a rigid material such as cement, plus the springiness of the clamp itself, ensures a continuous spring pressure during the setting period.

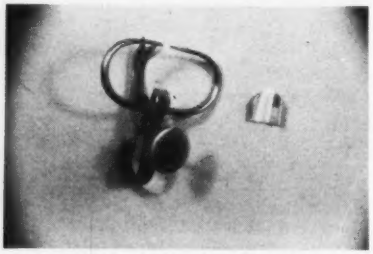


Fig. 3.—Hatch clamp with contoured aluminium.

For this reason, care is sometimes necessary to scrape the compound a little over the filling area before tinfoiling, so that the resultant filling will not be too flat or under-contoured.

(2) *Where the cavity extends under the free margin of the gum.*

In these cases, if the gum has not been previously packed back, it may be difficult to get a perfect margin by the foregoing method.

For this reason we use the "hatch" clamp, together with a piece of sheet aluminium (same source) carefully contoured to fit under the gum and well around the cavity, covering all the gingival half of the tooth (Fig. 3.).

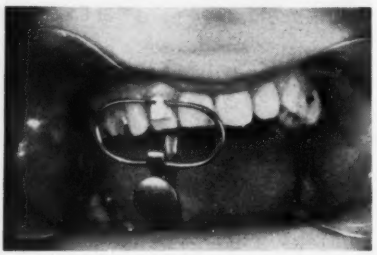


Fig. 4.—Hatch clamp in position.

Prepare the cavity and fill with the dough as before; now work the aluminium carefully into place and apply the clamp (Fig. 4.).

When the clamp has been tightened, take a small piece of stick compound (about ½") and soften one end in the flame, temper in air for about 10 or 15 seconds, and then press into place so as to cover the whole of the labial or buccal surface of the tooth.

This will aid in compression, and also prevent the clamp from slipping while the filling is setting.

CLASS III (PROXIMAL CAVITIES IN ANTERIOR TEETH NOT INVOLVING THE INCISAL EDGE).

The very small cavities in this class are not difficult to handle, but the large ones are. The more extensive the cavity, the more we come up against such problems as retention, contour, stresses of occlusion and shrinkage.

For this reason, use the utmost care in cavity preparation and in pressing the filling. If failures occur with early experience in the use of resins, it is in this class of cavity where they are most likely to occur; in fact, if the conditions look unfavourable, i.e., if there is a large shallow cavity with poor retention and an unfavourable bite, it might be advisable to consider some other filling material until considerable experience has been gained.

The sign of failure is, of course, the loosening of the filling. To be on the safe side, test all recently placed fillings with an explorer, pushing the filling in and out. If looseness is present, it can be seen as a slight movement at the

Fig. 5.—Metal strip cinched up with haemostats or artery forceps.

margin. Another sign is sensitiveness. Should the patient complain of this, be on the lookout for a loose filling.

Up to the time of writing, we have tried several methods of obtaining pressure in Class III cavities. The results so far do not quite come up to those in Class V cavities and a little more care must be exercised.

To facilitate compression, various strips of plastic or metal are now on the market, or in some cases thin copper or orthodontic band material may be used.

(1) *For small cavities having access from the labial,* proceed as follows:

(a) Thoroughly dry the cavity, using alcohol and warm air.

(b) Insert a strip so that both ends protrude from the labial, one on the mesial and one on the distal of the tooth to be filled. Pull taut and burnish the lingual.

(c) Now take a short piece of stick compound, heat one end and press it firmly against the lingual surface. This will stick everything in place, if the surface is not wet. The cavity is now ready to be filled from the labial, and when this is done—

(d) Take a pair of haemostats (artery forceps) or similar long-nosed pliers and seize both ends of the strip, so that the beaks lie vertically along the labial surface (Fig. 5.). A careful twist will then tighten the strip and compress the filling.

(e) Now take another short piece of stick compound $\frac{1}{2}$" long, heat one end and force it to position in the proximal space where the filling is, thus sticking the strip in place on the labial. For small fillings this is usually enough pressure but, for larger ones, a clamp is necessary.

(2) *For larger cavities*:

(a) After drying as before, take a short metal strip which is not too springy or stiff, place it so that one end is on the labial, and the other on the lingual. If access is from the labial, then burnish the lingual to proper contour. (If access is from the lingual, then the procedure would be reversed.)

(b) Take a square of aluminium $\frac{1}{2}$" to $\frac{3}{4}$", which has been covered with compound to a depth of at least $\frac{1}{4}$", soften and press to place on the lingual. The filling is now ready for insertion from the labial.

(c) When this has been done, carefully burnish the strip over the filling area, using a "beaver-tail" or some similar type of plastic instrument, taking care to preserve the proper contour and to have a snug fit along the gingival.

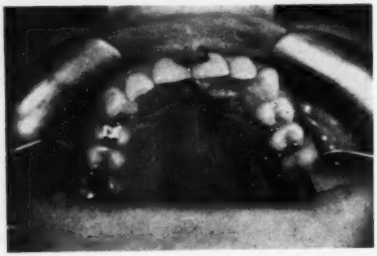


Fig. 6.—Showing metal strip burnished on labial and folded over.

(d) Now take another small square of aluminium with softened compound and press very gently to place.

The idea is to get the compound well into the embrasure without spoiling the contour. A little experience may be necessary before one finds out just exactly how much pressure to exert. It is also advisable to wait a minute or so before applying the clamp, with pressure brought to maximum in about a minute.

(3) *Filling two adjacent cavities such as two mesials in both central incisors*:

(a) In this case double the strip so that both ends are on the labial when it is inserted, leaving a small loop on the lingual (Fig. 6.); flatten and burnish the loop to the lingual of both teeth, covering the margins generously.

(b) Press the softened metal-backed compound to place on the lingual.

(c) Insert the filling from the labial and carefully bend and burnish each end of the strip over its corresponding filling.

(d) Now soften another impression, as previously described, and press gently to place and wait about a minute before applying the clamp as before. If there has been an excessive loss of either labial or lingual enamel in Class III cases, they had better be pressed as for Class IV cases.

CLASS IV CAVITIES (ANTERIORS INVOLVING THE INCISAL EDGE).

Only those cavities which do not show too great a loss of incisal enamel may be considered satisfactory; all others should be restored in some other way, such as by inlay with self-curing resin on the labial.

There is a definite indication, however, that Class IV cavities, when restored by self-curing resin, are likely to stand up better than when silicate is used.

(1) The method of choice so far in this class is by applying pressure similar to that in Class V cavities.

Fig. 7.—Metal-backed compound impression with and without tinfoil.

(a) Take the lingual impression first if access is from the labial, then press the labial impression to place making sure that both meet, and protect the incisal. Chill and remove; then carefully carve away enough compound in the filling area to leave an over-contoured filling. Tinfoil the filling area on both templates or impressions (Fig. 7.).

(b) Place the lingual impression in place, pack the filling, then carefully seat the labial and apply pressure with the clamp as before. This method, if carefully carried out, gives a well-condensed filling, but has the disadvantage

Fig. 8.—Copper band partly covering tooth.

of leaving excess filling material to be cleared away from the embrasure. This, however, is by no means enough to condemn it.

(2) This method consists of covering or partly covering the tooth by means of a copper band (Fig. 8.) or a plastic or celluloid crown form, to be stripped away when the acrylic has set. The method hardly needs explanation, except to say that we should choose as close-fitting a band or form as possible

and that the copper band should be burnished to the lingual and squeezed flat along the incisal edge. When this has been done and everything thoroughly dried, both the cavity and the band or form are filled to more than normal requirements and then pushed firmly to place. The small stick of compound may be used to tack it in place while setting.

It is obvious that this method could be used to fill both mesial and distal surfaces at the same time.

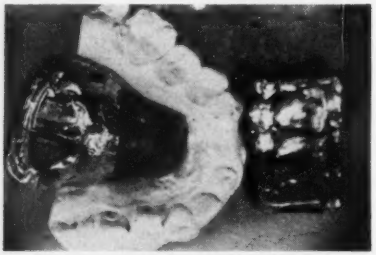


Fig. 9.—Model with tinfoil templates, ready to press temporary bridge.

Class I and Class II Cavities.

In the great majority of cases we are not concerned with aesthetics in these cavities but, if we are, then I believe it is better to cut away a part of an inlay or metal filling and fill only the visible part of the restoration with plastic.

If for some special reason complete Class I or Class II fillings in plastic are desired, then it is easy enough to surround the tooth with a matrix or

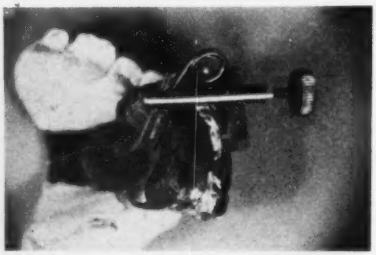


Fig. 10.—Clamp applied for pressing temporary anterior bridge.

copper band, using stick compound as a plunger to secure pressure during the setting period.

Other Uses.

Pontics and facings.

The replacement of a pontic or facing may be carried out by using the same technique as for Class V cavities. The part to be restored should first be waxed up to enable the necessary impressions to be taken and, in the case of a pontic of the "porcelain-tipped" kind, it may be necessary to protect the

gum with a soft metal strip passed from buccal or labial through to the lingual and fitting closely to the gum.

Temporary Bridges.

In case of accident, a patient with a broken porcelain bridge can be in a very embarrassing situation, and it usually takes time to make a temporary denture.

A temporary bridge could be made with self-curing resin in about an hour or two.

A stone model is made from an alginate or hydrocolloid impression, and the bridge is waxed up in ordinary pink wax. The labial and lingual impressions are taken as in the Class V technique. The wax is removed, and the model is then tin-foiled where necessary. The packing of the resin and clamping are routine. If the first one is not a success, several can be made off the one model, as the resin and tinfoil usually lift clear of the model.

Fig. 11.—Temporary bridge just after pressing.

These and similar uses will suggest themselves in all kinds of cases as they arise (Figs. 9-12.).

FINISHING.

The finishing and polishing is similar to that of denture resin. Care must be taken not to overheat and, if the resin is inclined to be slow-setting, it might be advisable to defer polishing until a later date. Brushes and cups, run at slow speed, charged with pumice or other polishing paste, will take care of most exposed surfaces. It is under the free margin of the gum, especially in the interproximal area, that the utmost care is necessary to get rid of the fine

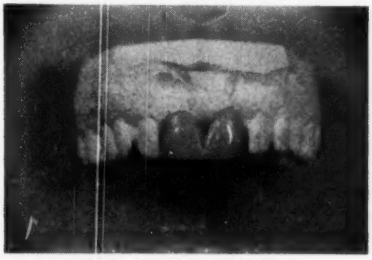


Fig. 12.—Finished temporary bridge.

feather edges of acrylic. These are almost invisible and it is necessary to dry with compressed air to aid examination.

The careful use of an old No. 700 bur will materially assist in removing gross excesses of material. This can be followed by a "lightning" or diamond strip, with a fine cuttlefish strip to finish.

Various knives with either a push or a pull action will be found useful in cutting away the last vestige of overhang; also, one may grind a "sickle" scaler so that it has a cutting edge to serve the same purpose.

Burs and stones may be used in a similar way to their use in finishing other filling materials, the round bur being particularly useful in contouring the lingual surfaces of anterior teeth.

Conclusion.

The use of self-curing resin in dentistry is a definite landmark in the progress of our profession.

While it still leaves much to be desired, it can fill a long felt want in our problem of aesthetics. Its manipulation requires care and patience, and there is no room for a "sloppy" technique.

If we remember that we should never expect a boy to do a man's job, then our judgment will be sharpened in deciding where to use it, and where not to use it.

THE CONSERVATIVE TREATMENT OF TEETH WITH EXPOSED PULPS IN CHILDREN

JULIEN REICH, M.D.Sc. (MELB.), L.D.S. (VIC.).

PATHOLOGICAL CONSIDERATIONS.

An exposed pulp must be regarded as infected, unless this occurred mechanically under strictly aseptic conditions. The infection may initially be confined to the surface of the pulp, but may penetrate to varying depth short of complete pulp involvement.

Since we may define inflammation as being the reaction of a *living* tissue to an injury, it follows that this state is present in varying degrees, in the exposed pulp. This may be due to:

1. Infection;
2. Mechanical injury resulting from instrumentation;
3. Chemical irritation, due to the use of drugs.

Inflammation of the dental pulp, as in the case of other tissues, is capable of resolution and, if not attended by cell death, will leave the pulp in a state indistinguishable from the normal.

If, however, some cells should die in the course of events, there will be an attempt to remove this necrotic debris, and to restore continuity by means of scar tissue, which may be more or less calcified[1]. Thus repair has been effected.

One of the possible factors which may prevent the removal of this necrotic cell debris is infection. The establishment of bacteria within this debris may be accompanied by the production of toxins which, on diffusing into the surrounding tissue, are quite capable of seriously interfering with the action of phagocytic cells in their attempt to remove the cell debris. It is conceivable that a state of adjustment in the host-parasite relationship could be established, resulting in the production of a chronic inflammation.

The importance of such nidi has been stressed by Fish[2], especially in relation to streptococci. These nidi may be present in the pulp in the form of dead pulp tissue, or particles of infected dentine pushed into the pulp at the time of exposure or instrumentation. Again, they may consist of the contents of dentinal tubules, whose respective cell bodies—the odontoblasts—have died as a result of injury.

In the case where the necrotic debris is not infected at the time of cell-death, it may nevertheless become infected in the interval between actual cell-death and removal of the dead cells by the phagocytic system. Such secondary infection of dead cells may be the result of:

(1) Communication with the oral cavity via the exposure.
(2) Lymphatic anastomosis between gingival margin and pulp.
(3) Bloodborne infection (bacteraemia)[2, 3].

Inflammation of the dental pulp may, of course, progress to complete necrosis of the tissue, being the result of strangulation of the apical vessels due to increase in the intra-pulpal pressure which accompanies the inflamma-

1. Fish, W. E.: Surgical Pathology of the Mouth; Pitman & Sons, London, 1948.
2. Fish, W. E.: The Distribution of Oral Streptococci in the Tissues; B.D.J., Sept. 15, 1936.
3. Gottlieb, Barron & Crook: Endodontia; C. V. Mosby Co., St. Louis, 1950.

tory process within the confined walls of the pulp canals. The process constitutes a progressive impairment of tissue nutrition accompanied by a lowering of tissue resistance (ability to react favourably to an injury), with the result that any infection present will tend to flare up with the production of an infected, dead, i.e., gangrenous pulp. It is therefore quite clear that we are dealing with a *dynamic process*, the exact extent of which we are unable to assess *clinically* by present-day means. Treatment, although based on sound principles, must of necessity remain empirical.

CLINICAL CONSIDERATIONS.

Choice of method.

When faced with an exposure of the pulp in teeth of children, the practitioner has the choice of one of the radical methods such as pulpectomy or extraction of the tooth; on the other hand he may decide in favour of the more conservative methods of pulp capping or pulpotomy.

In order to make his choice intelligently, he has to be quite clear as to the result to be aimed at. This may be said to comprise:

(1) Normal formation and resorption of the deciduous tooth-roots;
(2) Complete formation of the apices of permanent tooth-roots;
(3) Maintenance of archform and continuity, with consequent prevention of malocclusion;
(4) Aesthetic considerations;
(5) Lastly, but certainly not least, the physical well-being of the patient, from a medical point of view.

It is at once obvious that extraction of the tooth will not be in keeping with the first four aims listed above, although it is probably the only certain step to ensure the fifth aim. Pulpectomy, although satisfying our aims as regards prevention of malocclusion and aesthetic considerations, most decidedly falls short in regard to complete formation of the tooth apex and the medical future of the patient. The reason for this, of course, lies in the difficulty of filling pulp canals with open apices and of preventing periapical infection in complete pulp extirpation.

Turning to the conservative measures at our disposal, we find that pulp capping, which is essentially the covering of the exposed pulp surface with some suitable material, falls short of satisfying our aim as regards the medical future of the patient. This is, of course, quite obvious in the light of the remarks made above on the pathology of exposed pulps. Unless the pulp is exposed mechanically under strictly aseptic conditions, the capping of an exposure must be regarded as the deliberate enclosure of infection within the pulp chamber. The result of this may be an acute flare-up, or the production of a focus of infection which, although asymptomatic locally (and therefore regarded as successful!) may nevertheless be responsible for considerable future misery to the patient[4]. Certainly, an attempt may be made to sterilize the exposed pulp surface by means of some antiseptic, but the question immediately arises:

(1) Does the antiseptic act on the surface only, or does it penetrate the pulp tissue?
(2) What effect has it on the pulp tissue?

4. Stones, H. H.: Oral & Dental Diseases; E. & S. Livingstone, Edinburgh, 1948.

The issue is still an open one, thus one writer[5] ascribes quite a measure of success to the use of phenol on the exposed surface prior to capping. Thomas[6] found that phenol applied to cavities caused inflammatory reactions in the underlying pulp. This is supported by Manley's observation[7] that phenolizing of exposures prior to capping somehow interfered with the deposition of calcific material, and that chronic inflammation could be detected in these pulps after a period of 15 months.

Pulp capping, to say the least, is thus a very uncertain proposition and it is with this in mind that we pass on to a consideration of pulpotomy.

PULPOTOMY.

The rationale of this treatment is an attempt to remove the infected portion of the pulp, leaving behind the uninfected portion which, though certainly the seat of inflammation, could be expected to return to normal if:

(1) The cause of the inflammation is removed;

(2) The conditions are favourable, i.e., open apex, etc.

The question, of course, is how to determine the extent to which infection has progressed, and where to amputate the pulp. The answer, unfortunately, is that we cannot do so with present-day means. Strictly speaking this procedure differs from pulp capping only in that an *attempt* has been made to remove the infection, which is certainly the least thing we can be expected to do if we undertake to treat an exposed pulp. The search for more and better materials for covering the pulp tissue will not lead anywhere unless a method is developed which will enable us to determine the exact pathological state of the pulp.

Since pulpotomy aims at the removal of any infection present, at the same time retaining pulp vitality with its abovementioned advantages, we must regard it as the soundest of the methods available to us at the present time. It is with this in mind that this method will be discussed in detail. The operative steps are:

1. Clinical examination of tooth and gingival tissue.
2. Radiographic examination.
3. Determination of the state of the pulp.
4. Analgesia of the operative area.
5. Isolation and sterilization of field of operation.
6. Establishment of access to the pulp.
7. Removal of the coronal portion of the pulp.
8. Control of bleeding.
9. Application of the covering material.
10. Periodic checking of the case.

1. *Clinical examination.*

This comprises inspection of the tooth to determine the path of access and possible difficulties in restoring the crown. Occlusion should also be checked. A point of the greatest importance is to note the condition of the gingival tissue in relation to the tooth.

5. Rosenstein, S. N.: Pulp capping in Deciduous Teeth; J.A.D.A., 29:1632, 1942.
6. Thomas, B. O. A.: J.D.Res., 20:435, 1941.
7. Manley, E. B.: Proc.R., Soc. Med., 39:637, 1946.

2. *Radiographic examination.*

The importance of this lies in the information we may gain as regards:

a. Development of the root;

b. Shape of pulp chamber;

c. Degree of resorption or formation of the root;

d. Condition of the periapical region.

3. *Determination of the state of the pulp.*

As pointed out above, it is our inability to do just this which makes pulpotomy such a precarious undertaking. Although we cannot as yet determine the extent of the pathological process in the pulp, we have at our disposal certain means which may be used to determine whether or not pulpotomy is indicated.

First and foremost one should place the history of toothache. Thus, if there has been spontaneous toothache of more than momentary duration, it is safest to assume that the pulp involvement has progressed too far to allow for a successful separation of involved and non-involved portions[8]. Sensitivity to manipulation of the dentine is probably the best indication of vitality of the pulp from our point of view. Of course the possibility of a periodontitis must be kept in mind. If any doubt exists as to the vitality of the pulp, the operation must not be undertaken.

4. *Analgesia.*

This should be obtained either by infiltration, block or intra-osseous injection, depending on the tooth in question and preference of operator. It is sometimes wise to give the child as a pre-operative sedative one drachm of Elixir Nembutal.

Other methods of desensitizing the pulp such as the use of pastes, caustic drugs or arsenic, must be condemned. Our aim is to retain the vitality of the pulp remnant. It is impossible to control the application of these materials sufficiently to prevent endangering the whole pulp. Pressure analgesia must be condemned more strongly since it can only serve to push any infection present at the surface into deeper parts of the pulp, and is thus contrary to the very purpose of the operation.

5. *Isolation and sterilization of the field of operation.*

The aim of this step is:

(a) To reduce as far as possible the infection present on the surface of the exposure prior to instrumentation.

(b) To isolate and sterilize the adjacent area, so as to prevent contamination of the pulpotomy wound prior to the application of the covering material. It is, of course, obvious that all instruments used in this procedure must be sterilized.

Rubber dam should be applied where possible to isolate the tooth in question. Ivory clamp (2a) is especially useful to secure the dam to a deciduous molar. As regards the sterilization of the exposed surface, the writer is not aware of the existence of an antiseptic agent, which can be relied on to render the exposed infected pulp sterile, without impairing the vitality of the pulp.

8. Herbert, W. E.: B.D.J., 1945.

The use of such agents as hydrogen peroxide, Metaphen, Zephiran, must be regarded as empirical. The use of arsenic for this purpose, although quite effective from a theoretical point of view, must be dismissed because of our inability to control the extent of penetration. Similarly, cautery of the surface is an uncertain procedure, for although its application (and thus its penetration) is far more subject to our control, it inflicts a severe burn on the pulp, and thus increases the inflammatory reaction.

In order to determine the value of calcium hydroxide in this respect, the effect of this material on Streptococcus viridans *in vitro* was investigated, with negative results[9].

At the present time most workers report the use of Tinct. of Iodine, 2.5% though its justification for this purpose has not been proved.

6. *Access.*

Since the success of the operation depends largely on the complete removal of the involved portion of the pulp, it is obvious that the roof of the pulp chamber must be opened completely. Depending on the type of tooth concerned, this may be achieved by progressively burring the roof of the pulp-chamber till it is of paper thickness, and then completing the operation with a hand instrument. The important point to bear in mind is to prevent maceration and penetration of the pulp at this stage.

7. *Amputation of the Pulp.*

The method which appears to be the most popular is to use a large, round bur, which is rotated in the pulp chamber, thus more or less twisting the coronal portion of the pulp from the radicular portion. There are two objections to this procedure:

(a) It is conceivable that the use of a round bur may cause a twisting of the radicular portion thus endangering its vitality.

(b) The danger of penetrating the floor of the pulp chamber, even if minimal pressure is used with the bur. This is due to the very remarkable thinness of the inter-radicular portion of the pulp chamber floor, especially in lower second deciduous molars.

The use of small cutting knives, which may be constructed from old excavators, etc., has more to commend it. These should have a cutting edge, bent at a suitable angle, depending on the tooth concerned and the available access. They are passed along one of the proximate walls of the pulp chamber and, when the pulp chamber floor is reached, a mesio-distal cutting action is performed. The severed coronal portion may now be removed by withdrawing the knife or with the aid of a barbed broach.

The big danger in amputating the pulp is the possibility of pushing infection into the radicular portion during the introduction of the amputating instrument. The application of some electrosurgical appliance, capable of coagulating the contents of the pulp chamber without seriously affecting the contents of the radicular portion of the pulp, holds most promise in this direction.

8. *Control of Bleeding.*

Bleeding from the amputation wound may at times prove troublesome. This has been dealt with by the application of hydrogen peroxide, phenol, etc.

9. Bulate, Lydia: Personal communication.

In view of Manley's evidence that phenol may interfere with the deposition of calcific material[7], the use of this drug is not advocated. Zander found the application of sterile cottonwool pellets soaked in a saturated solution of calcium hydroxide quite effective[10]. Grossman recommends the use of a topical Thrombin preparation to effect immediate coagulation[11].

9. *Choice of the covering material.*

It is rather interesting to note the enormous amount of work that has been done in this field ever since the discovery that a vital pulp is capable of depositing or being the seat of deposition of calcific material. The aim of most investigators has been to find a substance capable of inducing the above reaction. To list the substances thus tried, with or without a just basis, would be to name every conceivable material that has passed through the hands of dentists.

On evaluating the various papers that have been published on the subject, one comes to the conclusion that two types of reaction can be seen in the pulp:

(a) The deposition of calcific material of no definite structure, related especially to any dentine fragments which may have penetrated the pulp during its exposure, or which may have been applied as a covering material.

(b) The deposition of a material closely resembling dentine in that it possesses tubules as well as a layer of cells resembling odontoblasts between the dentine and the pulp tissue.

Regarding the deposition of calcific material around dentine particles, it does not seem to matter as to whether they are derived from the tooth in question or from some other source. According to Manley these particles may act as "organizers," i.e., they determine the differentiation of adjacent mesenchyme cells, causing them to assume an osteogenic function[7]. The whole process could be regarded in the same light as the action of a bone-graft in an area of bone-loss. This method of formation of calcific bridges is probably the explanation of their reported occurrence in pulps covered with such materials as zinc oxide and eugenol, etc., which in themselves could theoretically at least not induce such a change.

The deposition of dentine on the other hand is claimed by a number of workers, notably Zander. He used calcium hydroxide, and considers that the essential factor seems to be the production of an alkaline reaction in the area.

Calcification in tissues, probably depends on the activity of the enzyme phosphatase, as well as on changes in the tissues themselves.

Blood and tissue fluid contain calcium and phosphate ions, actually in a state of supersaturation[12]. Furthermore, there are present in the blood and tissue non-ionized phosphoric acid esters. The enzyme phosphatase is capable of hydrolysing these into ionized inorganic phosphorous, thus causing a local increase in the concentration of the phosphate ions, above the critical solubility value. Now certain tissues in the body, especially the kidney, are rich in phosphatase, but are not normally the seat of calcification. The explanation for this lies in the normal acidity of the tissue. Phosphatase requires an alkaline medium. This is normally provided in calcifying areas by the activity

10. Zander, H. A.: Reaction of Pulp to Calcium Hydroxide; J.D.Res., 18:373, 1939.
11. Grossman: Indian Dent. Review, Dec., 1948.
12. Best and Taylor: Physiological Basis of Medical Practice; Ed. 3, Williams & Wilkins, Baltimore.

of certain cells (osteo- and odonto-blasts). It seems clear, then, that phosphatase is essential in this train of events, and the question arises as to its origin. Phosphatase is, of course, known to be present in small amounts at least in all connective tissues. However, for its action in this process it appears to be required in greater quantity or capable of more effective action. It is probable that calcium hydroxide, due to its alkaline reaction, increases the effectiveness of the enzyme.

Provided then that the operation is performed under aseptic conditions, there seems little justification for the use of antiseptics and other irritants in the covering materials. It is of interest to note that even zinc oxide eugenol paste is capable of inducing a chronic inflammatory reaction in the pulp stump, and thus interfering with the healing of the amputation wound[13].

The autoclaved calcium hydroxide crystals may either be mixed with distilled water or insufflated as such on to the pulpotomy wound, where it will form a sticky mass with the exudate. A thin layer of paraffin wax or neutral varnish should be placed on top of the $Ca(OH)_2$, and this in turn sealed with $ZnPO_4$ or similar cement. The purpose of the wax layer, of course, is to prevent interaction of the alkaline calcium hydroxide and the acid-sealing cement.

In a recent article[14] Gardner advocates the use of a paste consisting of zinc oxide powder and saturated calcium hydroxide liquid. He claims the formation of a compound ($CaZnO_2$) in a reaction which involves the assumption of acidic properties by the amphoteric zinc oxide when brought in contact with the basic calcium hydroxide. No histological evidence is offered nor is an explanation given as to the advantage of this compound over the ordinary calcium hydroxide.

A point worth mentioning in relation to pulpotomies performed on incompletely formed permanent anterior teeth is that the sealing cement should rest on an extension of the cavity floor past the circumference of the root canal. This will act as a support and prevent pressure on the pulp stump. The patient should now be dismissed with instructions to return immediately should there be any discomfort. If not, the case is checked in two months' time. Pulp-testing is obviously useless due to absence of pulp from the coronal portion of the tooth. Instead we must rely on the history and on radiographic examination. The latter affords evidence of vitality if it discloses:

(a) Continued root formation in permanent teeth;

(b) Continued root resorption in deciduous teeth.

These observations are of far more value than the mere watching for the appearance of radio-opacities under the capping material, which seem to be generally regarded as evidence for the presence of calcific bridges.

The tooth must be kept under observation for as long as the patient is in the care of the dentist concerned.

In conclusion, one can say that the practice of pulpotomy merits a place in the field of preventive dentistry, in that it may postpone or dispense with the need for orthodontic or prosthetic treatment; but it may conceivably become an etiological factor of medical condition.

It is for this reason that the operation must be approached intelligently, and undertaken only after proper assessment of all available information.

13. Zander, H. A., & Glass, R. L.: Pulp Healing; J.D.Res., 28:97, 1949.
14. Gardner, A. F.: Partial Pulpectomy; O.S., O.M., & O.P., 3:498, 1950.

INTRA-OSSEOUS ANAESTHESIA *

R. M. KIRKPATRICK, D.D.Sc. (SYD.).

One of the fundamental preliminaries for good operative or surgical work is perfect anaesthesia; the operative or surgical service then becomes a pleasure to perform.

No single injection technique at our command can assure us of this state of anaesthesia on all occasions and, in consequence, we need an expert knowledge of every anaesthesia technique to assist us. The method assuring the best chances of success is usually the intra-osseous technique.

In this technique we inject the solution into the cancellous bone as near the apex of the tooth as possible, through a reamed-out channel traversing the gum and cortical layer of bone.

This method, however, has some disadvantages and I do not advocate its use as a routine procedure for obtaining anaesthesia of the molar teeth. This is because I think the tuberosity (or the posterior superior alveolar) nerve block is easier in the case of the upper molars and the mandibular nerve block is easier for the lower molars although the anaesthesia is not always as deep as one would like it to be.

The main advantages of intra-osseous technique are that we more easily and more quickly obtain a profound depth of anaesthesia enabling pulp removal if necessary; there is no distension of the tissues or feeling of swelling and the numbness does not extend over a large area of the soft tissues such as the lips or cheek.

The main disadvantages are that of discomfort to the patient while the anaesthetic solution is being injected (this is due to an increased heart-beat rate which can be distressing to some patients); the shorter period of anaesthesia; the difficulty of penetrating into cancellous bone at times, especially if radiographs are not available; the danger of breaking reamers or needles and post-injection soreness if the interdental papilla has been penetrated by the reamer. Perhaps the use of the syringe twice on each occasion and the prolonged period needed to inject the solution may be disconcerting to the patient.

There are two common ways of obtaining anaesthesia for the teeth: either by depositing the anaesthetic solution around a nerve trunk and blocking it, or by infiltration of the solution into the area of operation.

Intra-osseous anaesthesia comes under the heading of infiltration anaesthesia. In the intra-osseous method we cause the solution to flow to the tooth apex through an artificially reamed foramen in the bone instead of through the natural foramina of the bone.

In the usual infiltration method of anaesthesia (supraperiosteal) a large amount of solution is deposited in the soft tissues near the bone overlying the tooth apex. This causes a ballooning of the tissues and compresses the solution. It is an advantage to deposit the solution beneath the periosteum (subperiosteally) so that it may compress and penetrate more easily. Providing the bone is porous it soaks, or runs under a slight pressure, through the canaliculi perforating the outer cortical layer of bone, to the tooth apex. When

*Read at the Twelfth Australian Dental Congress.

sufficient concentration has reached the apex, the pulp is anaesthetized and this lasts for some considerable time due to the continuous seepage of solution from the soft tissue reservoir. When this is exhausted the anaesthesia continues till it is washed out of the cells. In intra-osseous anaesthesia we have no reservoir of solution to keep seeping into the operative area so the period of anaesthesia is less; but of course it is quite easy to inject more solution again into the reamed-out channel.

In the other common methods of infiltration either the solution is deposited under pressure into the fibrous gingival papilla or the needle is inserted into the bony septum itself and the injection made (intra-septal anaesthesia).

In these instances the tissue cannot swell or balloon up. The solution is therefore forced into the deeper inter-septal bone and some travels right along the bony structure to the root apex. In these cases the anaesthesia is immediate and similar to intra-osseous anaesthesia. Neither of these methods of infiltration can be relied upon. In the first case the bone may not be sufficiently porous to allow the anaesthetic solution to flow through. In the second case the gingival margin may not be normal or sufficiently hard and fibrous to seal the needle in order that the anaesthetic solution may be forced through the inter-septal bone, or perhaps the alveolar crest is too dense to allow the solution or the needle to pass through.

In either case anaesthesia is not obtained. If, however, we can penetrate the mucous membrane, the periosteum, and the hard cortical layer of bone, and deposit our solution into the soft cancellous bone, the solution flows to the tooth apex with greater certainty and better concentration. In fact, the weakest of anaesthetic solutions are sufficient to produce profound anaesthesia. Since the bony walls cannot swell, on injecting the solution the anaesthetic travels through the bone rapidly with the slightest pressure. This creates pain unless the flow is extremely slow, especially to begin with.

It would seem that the blood-vessel walls in the bony structure are more penetrable by the anaesthetic solution, for the heart is affected by the adrenalin content after the deposition of the first drop or two. An intra-osseous injection therefore is somewhat similar in its systemic effects to an intravenous injection.

When contemplating an intra-osseous injection one must bear in mind the following principles:—

1. The nearer the injection is made to the tooth apex the better.
2. The thinner the cortical layer of bone the easier and safer it is to ream.
3. The soft tissue through which the reamer passes must not be flexible.
4. It is generally better but not essential to inject on the distal aspect of the tooth for positive anaesthesia.

A radiograph of the operative area is a tremendous help in deciding where an injection should best be made. The radiograph will show where the wider areas of cancellous bone exist and where a penetration can be made with certainty. It is indeed difficult to be certain of reaching cancellous bone in a confined area between the roots of two teeth which slant towards each other.

Bearing in mind the above principles, the reamer penetration when dealing with the upper teeth is best made at the intersection of two lines: one is

the horizontal muco-buccal fold where the soft flexible tissue meets the dense gingival tissue and the other is the vertical groove or crease in the tissue midway between the roots of the teeth. This groove is more easily seen when a coloured sterilizing solution is applied. It is not, however, always present.

In the lower jaw the cortical bone is too dense at the muco-buccal fold and it is therefore safer to attempt the penetration about 2 mm. above this line. The direction of the reamer when making the penetration should be either at right angles to the gum surface or, better still, slightly towards the apices of the teeth.

The only areas not constantly suitable for injection are the midline of the upper and lower jaws.

Any engine root canal reamer which is slightly larger in diameter to that of the syringe needle is satisfactory.

Suitable drills that are on the market at present are the Beutelrock No. 4 or No. 5 and the Swiss engine reamers, sizes 5 and 6. A long reamer that is too flexible can be cut short and the end stoned to a chisel edge. Reamers that have been used many times can be sharpened time and again but it must be remembered that the sides of the reamer are also meant to cut but the sides cannot be re-sharpened. A reamer should therefore be discarded after being used 100 to 150 times.

It is advisable to use the reamers in the straight handpiece. By so doing, one is confident that the syringe needle will follow into the reamed-out channel. The depth the reamer sinks into tissue on making a penetration varies from four to eleven millimetres, the most common depth measurement being six millimetres.

The site of penetration must first be anaesthetized by infiltration. In the upper jaw I insert the needle into the muco-buccal fold for about 2 mms. only and inject about one-third of a carpule of anaesthetic. In the lower jaw I insert the needle one or two millimetres above the muco-buccal fold, directing the needle towards the crown of the tooth. This last injection is more painful but it is more effective for the lower jaw. If a cocaine solution is favoured, inject only about half the volume or a little more for the same effect.

While the preliminary injection is infiltrating, I change the needle and hub, using a short needle (1 inch) and a long hub.

The mounted needle has previously been inserted into the centre of a disc of rubber one-sixteenth of an inch thick and about one-eighth of an inch in diameter. This acts as a sealing washer during the intra-osseous injection and also obviates a sharp bend in the needle, should the patient suddenly move his head. If the reamer channel is not deep enough for the insertion of the full length of the needle, a pair of cotton wool tweezers can be used to compress the rubber against the gum and the sides of the needle. This prevents leakage of the solution during the injection.

The reamer which is kept in Metaphen Disinfecting Solution is placed in the boiling water sterilizer for a minute or less and then inserted in the handpiece. A sterile cotton roll is used to keep the area of penetration dry and Metaphen is rubbed over the gum area. Careful observation is made for the point of penetration and the reamer rotating at slow speed is applied. Gentle, controlled pressure is used to ream through the cortical plate of bone and then the reamer will feel as though it drops into space as it reaches the soft

cancellous bone. The reamer is kept rotating while being withdrawn. Blood will immediately ooze from the puncture. The syringe is picked up, the blood is soaked up by the cotton roll, and the needle is immediately inserted into the reamer channel. It will generally sink readily down to the rubber washer. A little pressure on the syringe will seal the washer against the gum and the intra-osseous injection is then commenced.

The very first minim or two of solution injected immediately affects the heart-beat. In fact, by watching the patient's neck and observing the heart-beat pulsations one can be certain whether the solution is flowing intra-osseously or not. If the heart-beat is seen to increase, the anaesthetic solution is flowing through the bone and on completion of a correct dosage anaesthesia can be expected. Watch the patient's neck for an increase in the pulse rate and pulse volume. The pulse itself can often be felt in the lip or the external maxillary artery can be palpated as it passes over the inferior border of the mandible. If the heart pounds too heavily after the injection of 1 or 2 drops of solution it is necessary to wait a few seconds before injecting more. It should take about 10 seconds to deposit the first drop, about 7 seconds for the second drop and then, if the heart-beat is not too heavy, the flow may be increased to about 1 drop per 5 seconds. After 5 minims have been deposited, the rate can be increased to about 1 drop per 2 or 3 seconds. Generally 10 or 12 minims are sufficient to reach the apex but the effect would be transitory, were not another 5 minims deposited to form a pool of solution around the apex. I therefore inject 15 minims for anterior teeth but the canines may require 17 or 20 minims. Molars need up to 25 drops or more at times. Test for anaesthesia and, if not present, inject 50 per cent. more solution. If anaesthesia wears off during cavity preparation inject a half-dose again.

During the injection the patient may be told that it is the same anaesthetic that is being used but, due to the adrenalin content, his heart may beat heavily for about three minutes.

The injection must be made extremely slowly at first and never rapidly at any time. Remember the first five minims injected should take about 30 seconds and the remaining ten minims about another 30 seconds.

Test for anaesthesia by drilling or tapping the tooth. Usually anaesthesia is complete immediately after the injection but sometimes one may have to wait for a minute. If satisfactory, remove cotton roll and allow patient to rinse out. Anaesthesia may be expected to last for about 15 minutes but more solution can be injected into the same channel several times if necessary.

On dismissing the patient after the operation tell him not to brush his teeth in that area that day.

Providing the interdental papilla has not been reamed through, no after-pain need be expected.

In conclusion, may I suggest some practical tips in intra-osseous anaesthesia. Inject if convenient on the side of the tooth that is affected by caries, because the adjacent tooth generally needs attention and will be found to be anaesthetized. In the lower jaw inject on the distal side of a tooth if possible to insure anaesthesia; often the mandibular nerve is blocked by the injection and the teeth anterior to the side are also anaesthetized. A useful

site of injection is between the upper first molar and second bicuspid teeth. The molar and the two bicuspids are generally anaesthetized. An injection between the upper lateral incisor and canine will often cover the canine, lateral and central tooth. Inject between the roots of adjacent teeth if possible, as the roots guide the anaesthetic solution to the tooth apex. Do not ream too deeply between the lower incisors because the reamer may penetrate to the lingual tissues.

Remember the solution will flow in the direction of least resistance, which means it will flow out through the bony walls of a recent extraction socket or it will flow into soft cancellous areas rather than into the denser bone to be seen around a tooth apex at times.

When extracting teeth or removing deep-seated granulomata, make sure the area is well soaked with anaesthtic solution because a second intra-osseous injection after the cancellous bony structure is exposed is often ineffective.

The deciduous teeth are anaesthetized as readily as the permanent teeth. Injections can be made between the roots of a tooth or on the lingual or palatal sides if necessary. Be mindful of the possible presence of pus at the apex of a tooth in an acute condition. Do not disseminate the infection by injecting into the area.

Even if used only occasionally, intra-osseous injections can be marvellous reputation-maintainers.

The DENTAL JOURNAL of AUSTRALIA
A.D.A. N.S.W.

EDITORIAL DEPARTMENT

THE SUGAR MEN

The Colonial Sugar Refining Company has a valuable adjunct providing information service to its staff through a News Letter in which is presented a wide variety of material for its readers.

Naturally, it adopts a policy best suited to its own interests. It is a pity, however, when a phrase used for its dramatic euphemism by one of our leaders in preventive dentistry should be seized upon and its application twisted in such a manner as to detract from the well-known dangers of excessive intake of refined carbohydrates in the diet.

If Dr. Hearman relentlessly maintains his position in relation to sugar (and I do not believe that this is his attitude for one moment), I would remind the sugar men that they are more than blindly wedded to the Pennsylvania mass studies in human nutrition.

This well-worn cab-horse is whipped out of the stables at the least provocation, whereas one need only consult the Bowral experiment carried out by Bailey with a number of war orphans to observe the effect of a dietary consisting of protective foods with the almost complete elimination of sugar and to learn the value of proper dietary as a factor in health.

To use phrases such as "It is widely recognised that nations with a high standard of living have a high rate of sugar consumption; they have more dental caries; they have more telephones and motor-cars. If one can say that eating sugar causes dental caries, cannot one with equal logic say that talking on telephones causes dental caries? Or riding in motor-cars causes dental caries?" and describe this as logic simply places the remainder of the statement in its correct category—a scrappy attempt by an uninformed writer to oppose a stand made in the interests of the health of the people and recognised as a correct stand by a large number of nutritionists and experts on diet and health and diet in relation to disease throughout the world.

If progress is to be achieved in the solution of one of the greatest social problems to be met by present-day civilization, then the co-operation of all interests, particularly the food industries, is essential.

The Plenary Session on Dental Caries at the Twelfth Australian Dental Congress was designed to arouse the interest of the general practitioner in

the magnitude of the problem and in the current advances made towards its solution both in the laboratory and in the clinical field.

Hockett[1] contributed to a symposium on Sugar and Dental Caries at the Eightieth Annual Meeting of the California State Dental Association held at San Francisco in April, 1950, and strove seriously to demonstrate that sucrose as occurring naturally is identical to sucrose as a refined product. Of course, this cannot be denied but the "sugar" as taken in the spoon from the bowl is vastly different from the "sugar" as eaten in the orange or, for that matter, in the sugar-cane.

Hockett was anxious to demonstrate the caloric yield per acre of foodstuff to the immense advantage of the carbohydrate and there is none who would quibble with this contention, but there is no need to take the carbohydrate as sugar to the distinct detriment of one's appetite.

Naturally, the sugar interests wish to maintain and increase their production and profits and, if what Hockett says is true (again I quote): "There are some jokers in the statistics. It is necessary to be a bit careful in their use. Furthermore, even where *per capita* distribution of sugar may reach 100 or 103 pounds we know there are a number of corrections which must be made for non-food uses, for instance, all the sugar which is used in silvering mirrors must be deducted and even most of the sugar used in baking of bread, which is the biggest industrial utilization of sugar, because a great deal of the sugar is converted into carbon dioxide and like industrial alcohol is not consumed. Likewise much of the sugar going into the processing of cigarette tobacco or curing of meat is never consumed as sugar. Therefore, the figures must be used with some caution," the consumption orally of sugar is not nearly so high as the figures indicate, i.e., if we take Hockett's contention as serious (and he must be given due credit for his knowledge of the use of sugar in his own field).

It is therefore important to remember this when dealing with the question of the effect of sugar on the teeth when taken orally for, if the consumption of sugar *per capita* of the population includes these amounts used for the various industrial matters which Hockett has listed, then because of the smaller amount available for the food taken through the mouth, the influence of any rise or fall in that sugar intake is likely to become much more significant, and it is important to remember this factor when dealing with contentions raised by the sugar interests.

It seems clear that there are many fields for their own activities in industrial matters without decrying the efforts of the profession to reduce, not necessarily eliminate but reduce, the *per capita* oral consumption of sugar in foodstuffs when it is so readily demonstrated that, with the presence of sugars, particularly taken between meals in the form of beverages and sweets, caries becomes rampant where the particular organisms are known to be present in the mouth.

Assuming that sugar does not rot teeth, words Dr. Hearman has been so accused of using carelessly, it does something which is far worse—it unfortun-

1. Scientific Director, Sugar Research Foundation, Inc.; associate Professor of Chemistry, Massachusetts Institute of Technology.

ately acts as a strong diluting agent in modern diet. Because it supplies the calories, it reduces the desire of the person to eat those foods which are protective and necessary in building a healthy body and combating disease. Whilst it supplies the necessary calories, it is completely devoid of essential proteins, vitamins and minerals and so the more sugar consumed the less opportunity for eating the essential nutrients.

This far greater danger added to those already met by the present representatives of the human race in their constant warfare to gain from the earth's surface the essentials of life is a factor which the sugar men need to remember.

Correspondence

"BLACK TONGUE"

Sir,

With regard to the Case Report on this condition by H. A. Mueller in the August issue of the Journal, I would like to add these comments.

Black Tongue or Black Hairy Tongue is reported from time to time in medical and dental literature. I have references dating back to 1854.

A dental colleague referred a case to me about twelve months ago for advice as to treatment. The patient, a man aged 48 years, had been unaware of the condition until my dental colleague drew his attention to it. This was about 2½ years before I saw him. He worried about it and tried various treatments suggested to him by medical practitioners without success.

I advised him to clean the dorsum of his tongue after each meal with the toothbrush, keeping the brush well moistened with a solution of sodium bicarbonate (1 teaspoonful to a glass of water). He 'phoned me a fortnight later to say that he was very happy with the treatment. The condition had cleared up after three days of the treatment; but he noticed that it tended to recur if he left off treatment.

I shall not go into the rationale of the treatment; but I shall leave it to your readers to think out for themselves, always remembering that the mucin of the saliva is precipitated by acids, that there are bacteria in the mouth which produce acids, that sodium bicarbonate neutralizes acids and is also a solvent of mucin.

Yours faithfully,

ROLAND G. MORRIS.

147 Collins Street, Melbourne.
November 14, 1950.

News and Notes

ST. GEORGE DENTAL ASSOCIATION

Annual Meeting

The 27th Annual Meeting of this Association was held on 17th August, 1950, and was very well attended by members and representatives of the Western, Eastern and Northern Suburbs Dental Groups.

The election of office-bearers for the year 1950-51 resulted as follows:—

President: Mr. A. S. Binns.

Vice-President: Mr. E. Morris.

Hon. Secretary & Treasurer: Mr. N. E. Edney.

Sports Day

Roselands Golf Club was the venue for the Annual Sports Day; unfortunately, the bowls events had to be cancelled owing to rain. The results of the golf competition were as follows:—

Par handicap:	
Winner:	A. French.
Runner up:	T. Purtell.
Best 1st nine:	O. D. Oddy.
Best 2nd nine:	E. McGovern.
4-Ball Best Ball:	
1st:	A. French & T. Purtell.
Runners up:	H. Hicks & O. Stenmark.

POSITION VACANT

Eastman Dental Hospital and Institute of Dental Surgery (University of London)

Applications are invited from Dental Practitioners for the post of Junior Clinical Assistant for a period of one year from 2nd April or 1st October, 1951. Holders of these posts will have the privilege of attending all the lectures and demonstrations arranged by the postgraduate Institute of Dental Surgery and the Royal College of Surgeons of England for candidates preparing for the Primary and Final examinations for the Fellowship in Dental Surgery of the Royal College of Surgeons of England.

To be eligible for appointment, applicants' names must be on the Dentists' Register of the Dental Board of the United Kingdom. Preference will be given to persons who have held a previous House appointment. Salary: £500 per annum. Applications should be made, before 1st February or 1st August, 1951, to the Director of the Hospital, Gray's Inn Road, London, W.C.1, stating qualifications and subsequent appointments together with the names of two referees.

POSTGRADUATE COURSE

Institute of Dental Surgery (University of London), Eastman Dental Hospital.

A postgraduate course of nine months' duration, intended primarily for candidates preparing for the Primary and Final examinations of the Royal College of Surgeons of England for the Fellowship in Dental Surgery, will commence on 2nd April and on 1st October, 1951. In addition to clinical practice at the Eastman Dental Hospital, which will cover all aspects of Dental Surgery, the course will include lectures and demonstrations given at the Royal College of Surgeons of England in Anatomy, Physiology and Pathology and at the Institute of Dental Surgery in dental subjects. The fee for the course will be £60. The class will be limited in number and applicants should apply, before 1st February or 1st August, 1951, to the Dean of the Institute, Eastman Dental Hospital, Gray's Inn Road, London, W.C.1, stating qualifications and subsequent appointments.

FLAVELLE CUP

The competition for this golf trophy was held at Concord Golf Club on Thursday, 19th October, 1950, and in spite of the unfavourable weather 100 people took part. The winner was T. T. Dobson, who was also successful in 1935 and 1937.

After a very enjoyable day the presentations to the successful golfers were made by Mr. F. R. Reid and Mr. T. T. Dobson.

The results were as follows:—

Flavelle Cup:	T. Dobson.
Runner Up:	V. Golden.
Best Scratch Score:	E. King.
Country Trophy:	E. McGovern.
Handicaps:—	
12 and under:	I. Steele.
13—15:	J. Newman.
16—18:	A. Bull.
4-Ball Best Ball:	
1st:	T. Dobson and F. Holt.
Runners Up:	B. Smith and S. M. Hicks.
Consolation Prize:	W. A. Mitchell.

HARDWICK MEMORIAL LIBRARY

The following books have been added to the Hardwick Memorial Library since the previous list of additions was published in August, 1949:—

*American Dental Association.—Index to dental literature, 1945-47. 1949.

American Dental Association.—Horace Wells, Dentist: Father of surgical anaesthesia. 1948.

Brodsky, R. H.—Atlas of oral and facial lesions. 1948.

Campbell, J. Menzies.—A dental bibliography. 1949.

*Indicates reference books only.

Council on Dental Therapeutics (American Dental Association).—Accepted dental remedies. 1950.
*C.S.I.R.O.—Guide to authors. 1949.
Darcissac, Dr. Marcel.—Le bridge "amorti." 1949.
Drummond-Jackson, S. L.—Dental practice management. 1949.
Ericsson, Y.—Enamel-apatite solubility. 1949.
Fish, E. Wilfred.—Surgical pathology of the mouth. 1948.
Ginn, J. T.—Review of dentistry. 1949.
Goldman, Henry M.—Periodontia. 1949.
Gottlieb, Barron & Crook.—Endodontia. 1950.
Grossman, Louis I.—Lippincott's handbook to dental practice. 1948.
Kronfeld, Rudolf.—Histopathology of the teeth. 1939 (3rd Ed.).
Landa, J. S.—Practical full denture prosthesis. 1947.
Leicester, H. M.—Biochemistry of the teeth. 1949.
Levy, I. R.—Acrylic inlays, crowns and bridges. 1950.
Levy, I. R.—Textbook for dental assistants. 1948.
Manley, E. B., & Brain, E. B.—An atlas of dental histology. 1947.
Moon, G. R.—How to become a doctor. 1949.
Morrison, G. A.—In the dentist's office. 1948.
Osborne, John.—Dental mechanics for students. 1948.
Poldy, John J.—Australian dental practice in its relation to public health. 1946.
*Proceedings of the 11th Australian Dental Congress.
Sicher, H.—Oral anatomy. 1949.
Smith & Martin.—Zinsser's textbook of bacteriology. 1948 (9th Ed.).
Thoma, Kurt H.—Oral and dental diagnosis. 1949 (3rd Ed.).
Tylman, *et al.*—Year book of dentistry. 1949.
Trapozanno, V. R.—Review of dentistry. 1949.
Weinberger, B. W.—An introduction to the history of dentistry (2 Vols.). 1948.
Williams, H. E.—Humanizing our great profession. 1949.
Zeisz, R. C. & Nuckolls, J.—Dental anatomy. 1949.

*Indicates reference books only.

Association Activities

AUSTRALIAN DENTAL ASSOCIATION (NEW SOUTH WALES DIVISION)

Extract from the Minutes of the Meeting of the Executive Committee held in the Council Room, B.M.A. House, 135-137 Macquarie Street, Sydney, on Monday, 9th October, 1950, at 7.30 p.m.

Present: Dr. E. R. Magnus, President, in the Chair; Dr. F. E. Helmore, Vice-President; Dr. A. G. H. Lawes, Vice-President; Dr. R. M. Cloutier, Honorary Treasurer; Dr. J. V. Hall Best, Mr. H. McD. Finnie, Mr. W. A. Grainger, Mr. R. G. Leeder, Mr. J. W. Skinner, Mr. Ralph Tompson, Mr. R. W. Wilson, Mr. R. Y. Norton, Mr. F. R. Reid, Mr. R. Krauss; Dr. A. G. Rowell, Western Division; Dr. J. D. Oddy, South Coast Division; Mr. L. Mackenzie, Blue Mountains Division.

Apologies: Mr. N. E. Edney, Mr. H. R. Sullivan; Dr. J. D. Benson, North and North West Division.

In attendance: Mr. Robert Harris, Secretary.

Business Arising from Minutes:

Conference with representatives from Suburban Dental Groups: A letter from the Honorary Secretary of the Western Suburbs Dental Group was tabled, which stated that the opinion of this Group remains unchanged, i.e., "The Committee is unanimous in its opinion that such a conference, held twice a year, will be a good thing for maintaining close co-operation between the Groups and the A.D.A."

Dental Health—Insurance of Exhibit: The Secretary stated that he had arranged with Edward Lumley & Sons Ltd. to have this exhibit covered against damage. It was resolved that the Secretary's action be endorsed.

Australian Veterinary Association—Salaries of women graduates: The Secretary read a letter from this Association with which they had enclosed a copy of their Memorandum and Articles of Association. The letter stated *inter alia* that the A.V.A. considered the discrimination against women graduates inequitable. They were seeking the viewpoints of other professional Associations and, if it were considered that a change was desirable and could be effected by combined action, then further negotiations would be entered into.

It was resolved that a reply be sent indicating that we are in accord with the principle of equal pay for both men and women graduates.

Twelfth Australian Dental Congress:

Congress finance: Dr. Best stated that Congress finances had worked out in a very satisfactory manner. Final figures were not yet available as provision had had to be made for certain outstanding accounts.

A donation had been made to the University of Sydney and the United Dental Hospital. The sum of £260 would be paid to the Australian Dental Association (New South Wales Branch) as reimbursement for expenses incurred in connection with the organization of Congress, and it was a recommendation from the Honorary Officers that this amount be accepted as adequate.

Printing of Congress Proceedings: There had been many requests to have the proceedings of Congress printed as a bound volume. With this end in view Mr. Harris had gone most thoroughly into the matter of costs, and thanks were due to him for the trouble he had taken. Unfortunately, these costs were prohibitive and proceedings would therefore be printed in the Dental Journals.

It was resolved that the sum of £260 be accepted by this State Branch as adequate recompense for expenses incurred in connection with the organization of the Twelfth Australian Dental Congress.

Reports from Committees:

Dental Health Committee:

Propaganda: Mr. Tompson reported that the recommendations emanating from the Conjoint Meeting of the Executive Committee and Delegates from Divisions had been approved in full by the Dental Health Committee and that the Divisions had pledged themselves to carry out this work. The Dental Health Committee requested endorsement of the relevant resolution carried at the Conjoint Meeting.

It was resolved: (1) that a set of recordings be prepared by the Dental Health Committee for circulation among the Divisions who will then organize the broadcasting of same; (2) that the various Divisions appoint Committees at their next meetings to deal with the matters raised in this plan and that such Committees be authorized to go ahead with such items as were agreed to at the Conjoint Meeting.

Health Week Exhibit: Mr. Tompson reported that some expense would be incurred in connection with assembling, lighting and subsequent dismantling of the Dental Health Exhibit which was to be shown at the Town Hall during Health Week. He requested that an expenditure of approximately £15 be authorized for the purpose.

It was resolved that authority be given for an expenditure of not more than £20 for expenses in connection with this exhibit.

Staffing of Exhibit: Mr. Tompson raised the question of staffing the Dental Health Exhibit during Health Week beginning on 24th October, 1950, and stated that whilst his Committee were prepared to take duty at night he did not feel disposed to request that they do so during the working hours of the day. He asked that the State Branch Office supply an attendant to staff the exhibit during the day.

It was resolved that the Executive Committee approve in principle and that the Secretary make the necessary arrangements.

Cost of Essay Competition: Following the query raised at the August meeting of the Executive Committee regarding the cost of running the Dental Essay Competition, Mr. Tompson reported that on advice received from Miss Crook, who was handling the 1950 competition, the change in procedure had not greatly affected the cost involved. This year the Competition had been in the form of a quiz requiring detailed answers

whereas in former years it had been an essay and a simple quiz. He considered that either Miss Crook's charges were much higher than those of Buddee & Crawford, who had handled the 1949 Competition, or that rising costs were responsible.

Consideration was then given to the following resolution by the Dental Health Committe, viz.: "That this Committee is opposed to the practice of sending work associated with the Dental Essay Competition out of the office and considers that the work should be handled within the office itself."

It was resolved that the Secretary be instructed to investigate and report to the Executive on this proposal.

Newcastle Health Week: Referring to Newcastle Health Week, Mr. Tompson stated that seven of the Newcastle members had given lectures at the schools. The Dental Health Committee had received a letter from Mr. Mann, the Organizing Secretary of Health Week, speaking very highly of the part these members played in making the activities of Health Week successful. The Dental Health Committee wished to record their appreciation of the part played by the Newcastle members.

Divisions:

Mr. Krauss reported that it had been decided to hold the Convention at Armidale during the week 20th to 25th August, 1951, and requested the Committee's approval.

It was resolved that this Committee approves the holding of the Armidale Convention during the week 20th to 25th August, 1951.

Successful meetings to hear Dr. Terrell's lectures at Lismore, Wagga Wagga and Newcastle were reported by Dr. Magnus, Dr. Helmore, Mr. Krauss and Dr. Lawes.

Journal:

Mr. Tompson reported that the Journal Committee had met to consider the recommendation which had been made by the Conjoint Meeting of the Executives and Delegates from Divisions as follows:

"That it be recommended that the Journal Committee consider giving more items of general interest in the Dental Journal of Australia and not quite so many of the highly technical articles which have recently appeared."

The Journal Committee at its meeting on 27th September, 1950, had passed the following resolution:

"That it be recommended to the Executive Committee that, in view of the report made by the Editor concerning the diversity and quantity of material to be presented from the lectures and clinics of the Twelfth Australian Dental Congress and other sources, it is the opinion of the Journal Committee that the high standard of the Journal will be maintained and that no change in policy is recommended for the ensuing twelve months."

It was resolved that this recommendation be adopted.

Annie Praed Oration:

Dr. Magnus stated that it had been a recommendation from the Executive Committee that an Oration to be known as the "Annie Praed Oration" be established and given initially at the Annual General Meeting of the New South Wales Branch in 1950. The Twelfth Australian Dental Congress had prevented completion of the arrangements and he would recommend that the oration be omitted in the year when a Congress is held in New South Wales.

It was resolved that the Annie Praed Oration be omitted from the Annual General Meeting this year and in any year in which a Congress is held in this State. It was also resolved that the question of any fee to be paid to the lecturer be referred to the incoming Executive.

Membership:

New Members: It was resolved that the following dental practitioners whose applications were in order and who had paid the requisite subscriptions be admitted to membership of this State Branch as from 9th October, 1950:

Arthy, David Henry, B.D.S.; Hall, Ronald Graham, B.D.S.

Resignations: It was resolved that the resignations of Mr. Philip Davies, B.D.S. and Miss Ellen Joan Hall, B.D.S., be accepted as from 31st December, 1950.

Correspondence:

Australian Society of Orthodontists: A letter from the Secretary, Mr. R. Y. Norton, stating that this Society had been re-formed on the occasion of the Twelfth Australian Dental Congress as a specialist Orthodontic Society was received.

A.D.A. Certified Dental Materials: A letter from the Federal Office requesting that each State Branch initiate individually in its own State the action necessary to ensure that, where the Standards Association of Australia has adopted a dental standard, Government and semi-Government contractors should be obliged to certify that their products comply with this standard was received.

Alien dentists: A letter from the Federal Office, requesting that the State Branch prepare information as early as possible in regard to the registration in Australia of

overseas dentists who do not hold a qualification acceptable to the General Medical Council of Great Britain, was received.

It was resolved that the request of the Federal Office be carried out by this State Branch.

Western Suburbs Group: The Secretary read a letter from the Western Suburbs Group embodying four resolutions which had been passed at a meeting of delegates from the St. George Association and the Suburban Groups held at B.M.A. House on Thursday, 28th September, 1950.

It was resolved that the matters contained therein be referred to the next meeting of the Honorary Officers for report to the next meeting of the Executive Committee.

SOUTH COAST DIVISION

The Annual Meeting of the South Coast Division was held on Friday, 3rd November, 1950, at 8.30 p.m.

The Office-bearers elected for 1950-51 are as follows:—

President: Mr. Ashton Marshall.
Vice-President: Mr. R. G. Esdaile.
Hon. Secretary: Mr. J. H. Palmer.
Asst. Secretary: Dr. J. D. Oddy.
Hon. Treasurer: Mr. R. G. Esdaile.
Additional Member to Executive Committee: Dr. J. D. Oddy.
Chairman, Dental Health Committee: Dr. J. D. Oddy.
Sports Secretary: Dr. J. E. McGovern.

SOUTHERN TABLELANDS DIVISION

The election of Office-bearers for this Division for the year 1950-51 was held at the Annual Meeting on 4th November, 1950, and resulted as follows:—

President: Mr. Gordon Cattle, Goulburn.
Vice-President: Mr. L. Cooper, Queanbeyan.
Hon. Secretary-Treasurer: Mr. K. R. Fisher, Goulburn.
Executive Committee: Mr. M. Hamilton, Moss Vale; Mr. L. Marshall, Canberra; Dr. D. MacCulloch, Goulburn.
Divisional Representative: Mr. L. Cooper.

New Books and Publications

Oral and Facial Cancer, by B. G. Sarnat and I. Schour, Chicago, 1950. The Year Book Publishers, Inc. *Our copy by courtesy of the publishers.*

This book has been prepared as a result of American experience and practice and it is a most important advance to observe the importance and emphasis placed on the value of complete co-operation between the medical and dental professions.

The one great danger from carcinoma—insidious onset with early metastatic change—must constantly be kept before the profession and the public. The unfortunate experience, met by many patients whose untimely death could have been avoided, is the absence of a diagnosis at a stage when cure was certain.

It is stated that forty per cent. of all cancers of the head and neck are seen first by the dental surgeon; if this is so, then a heavy responsibility lies on

the profession, and the authors are to be commended for preparing this informative book.

The book is divided into three sections: Part I deals with the cancer problem on the basis of the public health aspects and present status of cancer research, and Part II is devoted to clinical aspects.

Part I therefore brings with forceful impact upon the reader the statistical analysis of the problem with its percentages of incidence and its lethal powers diagrammatically presented.

Part II takes the various areas of the head and neck and presents the clinical picture of lesions occurring in these regions. The text is amply supported by illustrative material from cases together with examples of precancerous lesions.

Part III is devoted to the control of the disease and considerable emphasis is laid upon the necessity of careful history and examination of each case. The importance of laboratory aids is given due support, though far too many practitioners are prone to examine superficially any problem which is presented and then transfer the patient to other hands.

Some really excellent clinical material is presented in the section on differential diagnosis of non-cancerous lesions and swellings of the neck. A general statement in some detail on the treatment by surgery and by prosthesis provides the dental surgeon with useful information not only for himself but for transmission to the unfortunate patient. For though we may have much skill as surgeons or prosthetists we are serving our fellows, and a careful phrase expressing a buoyant hope may change a patient's whole outlook on life.

The dentist's role in the care of the patient consists in developing a high standard of oral hygiene with a minimum of traumatic interference; in the field of pre- and post-surgical care, skilled prosthetic appliances are often necessary and the specialist in this field finds an interesting variety in his work.

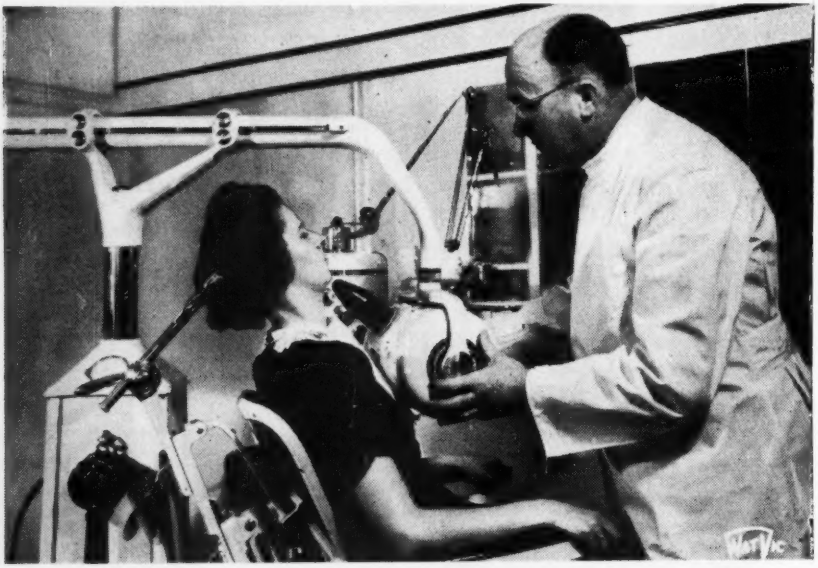
At the end of each Part a series of questions provides a useful means of recapitulation, whilst the Appendix reiterates some important points set out in detail in the book.—R.H.

The Dental Practitioner. Published by John Wright and Sons Ltd., Bristol.

This is a new monthly journal designed primarily for those engaged in the practice of dentistry. The editorial in the first issue states that the aim is to publish helpful matter in an interesting and pleasing manner; it is also planned to use illustrative material as much as possible.

This issue contains an article on roentgenology (bite-wing films), a review of local anaesthesia, a case report on mandibular asymmetry. In addition to a section devoted to hints on clinical practice, features are presented for the technician and nurse.

As a practical and informative publication, the *Dental Practitioner* should be well received.—R.H.



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